Transactions of The New York Academy of Sciences

SERIES II

VOLUME 22

NEW YORK

PUBLISHED BY THE ACADEMY

1959-1960

CONTENTS OF SERIES II, VOLUME 22

PAGE

Title page i
Contents ii
In Memoriam: Cornelius Packard Rhoads. *By* DAVID A. KARNOFSKY 3
Some Stratigraphic Problems of the Cow Head Area in Western Newfoundland. *By* C. H. KINDLE AND H. B. WHITTINGTON 7
Studies on the *in Vitro* Accumulative Process for Sulfate Ions of Mammalian Kidney Cortex. *By* INGRITH J. DEYRUP AND JEANETTE C. RODNAN 19
Energy Production from Nuclear Reactions. *By* HARRY SOODAK 26
The Use of an Analogue Computer for Measurement of Respiratory Depression. *By* WELDON BELLVILLE AND JOHN C. SEED 34
Enzymatic Autoinduction and the Hypothesis of Intracellular Permeability Barriers in *Neurospora*. *By* S. R. GROSS 44
Dowry and Inheritance in Modern Greece. *By* ERNESTINE FRIEDL 49
Conferences Held:
Freezing and Drying of Biological Materials 55
The Metabolism of Oral Tissues 57
Numerical Properties of Functions of More Than One Independent Variable 62
New Members 65, 150, 216, 290, 380, 479, 556, 697
The Genesis of Cancer and Possible Interference with its Growth. *By* ALEXANDER BERGLAS 83
The World-Wide IGY Cosmic Ray Program. *By* ROBERT C. HAYMES 96
In Vivo Dosimetry with Miniature Radioluminescent Glass Rods. *By* STANLEY J. MALSKY, CHARLES G. AMATO, BERNARD ROSWIT, CYPRIAN B. REID, S. M. UNGER, CHARLES SPRECKELS, AND EMANUEL VILLAZON 104
Kinship Organization Among the Urban Batak of Sumatra. *By* EDWARD M. BRUNER 118
A Study of the Effect of Weather on Mortality. *By* PAUL H. KUTSCHENREUTER 126
Conferences Held:
Modern Concepts of Thyroid Physiology 139
Care and Diseases of the Research Monkey 142
Automatic Chemical Analysis 145
Report of the Annual Meeting 157
Abstracts of 1959 Award Papers:
A Petrologic Investigation of the Manlius and Coeymans Limestones. *By* FRANKLIN W. FESSENDEN 173
Diffusion Behavior of Supersaturated Binary Solutions. *By* JACOB MAZUR AND TURNER ALFREY, JR. 174
The New York Academy of Sciences Awards for 1960 174
Viruses 1959. *By* HILARY KOPROWSKI 176
Some Observations on Structure and Metamorphism in the Harrisburg, N.Y. Quadrangle. *By* R. BRUCE THOMPSON, JR. 191
Methods, Problems, and Results of Growing Plant Cells Under Submerged Conditions. *By* WALTER TULECKE AND LOUIS G. NICKELL. 196
Conferences Held:
Nonnarcotic Drugs for the Relief of Pain and Their Mode of Action 207
Chelation Phenomena 210
The Reticuloendothelial System (RES) 213
Geological Problems in the Construction of Dams. *By* KEMBLE WIDMER 223
Nuclei Formation and Crystal Growth in Mineralizing Tissue. *By* ALBERT E. SOBEL, PENNI A. LAURENCE, AND MARTIN BURGER 233
Action of Lung Chemoreceptors on the Cardioinhibitory Center. *By* P. RUDOMIN Z. 244

Effect of Pentobarbital and Meperidine on the Central Respiratory Mechanisms in the Cat. *By* S. H. NGAI 252
Intelligence, Quantum Resonance, and Thinking Machines. *By* DAVID WECHSLER 259
Some Recent Developments in the Sociology of the BaMbuti Pygmies. *By* COLIN M. TURNBULL 267
Arcas High-Altitude Meteorological Rocketsonde System. *By* JOHN A. BROWN 275
Conferences Held:
Connective Tissue and Diseases of Connective Tissue 284
Second Conference on Medical Mycology 287
The Strength of the Earth's Crust. *By* WARREN CAREY 303
Physical Adsorption Processes and Their Relation to Order-Disorder Theory. *By* J. M. HONIG 313
Experimental Yellow Fever. *By* W. D. TIGERTT, T. O. BERGE, W. S. GOCHENOUR, C. A. GLEISER, W. C. EVELAND, C. VORDER BRUEGGE, AND H. F. SMETANA 323
The Interdependence Between Instrumental and Chemical Analytical Approaches. *By* SIDNEY SIGGIA 334
Slow Neutron Interactions with Matter. *By* T. J. KRIEGER 339
An Experimental Analysis of the Role of Context in Verbal Behavior. *By* CHARLES N. COFER 341
Tumors as Unique Biological Tools in Basic Biochemical Studies. *By* VERNON RILEY 348
Mycoparasitism of *Gonatobotryum Fuscum* and *Piptocephalis Xenophila*. *By* ALEX L. SHIGO 365
Conferences Held:
Fourth Tissue Homotransplantation Conference 373
Amino Acids, Peptides, and Proteins 377
Contributions of Stereoregulation to the Basic Principles of Polymerization. *By* NORMAN G. GAYLORD 387
Operational Control and Instrumentation in the Sugar Industry Today. *By* PIETER HONIG 399
The Danger of Simplified Ecosystems. *By* F. FRASER DARLING 408
Area Resource Development. *By* WALLACE BOWMAN 410
Culture Patterns and Resource Management. *By* PAUL B. SEARS 415
Radio Astonomy and the New National Observatory. *By* RICHARD M. EMBERSON 419
Properties of Heart Muscle Cytochromes Prepared with Deoxycholate. *By* WALTER W. WAINIO 426
Studies on Immunity to Coccidioidomycosis Induced in Mice by Purified Spherule, Arthrospore, and Mycelial Vaccines. *By* H. B. LEVINE. 436
The Changing Roles of Women in Suburbia. *By* NANETTE E. SCOFIELD 450
Meteorology: A Rediscovered Scientific Frontier. *By* THOMAS F. MALONE 458
Conferences Held:
Second Conference on the Mechanisms of Cell Division 462
Geochronology of Rock Systems 465
Aspects of Insect Endocrinology 470
Cardiovascular Effects of Nicotine and Smoking 471
The Actinomycins and Their Importance in the Treatment of Tumors in Animals and Man 476
Notice to Active Members 488
Pleistocene Climate. *By* WILLIAM L. DONN 491

Experiments on White Pekin Ducks Injected with DNA from Khaki Campbell Ducks. *By* J. BENOIT, P. LEROY, R. VENDRELY, C. VENDRELY 494
Cellular Identities in the Pars Distalis of the Rat Pituitary. *By* J. D. LEVER AND R. R. PETERSON 504
Pupillary Reactivity as a Measure of Autonomic Balance in the Study of Psychotic Behavior: A Rational Approach to Chemotherapy. *By* LEONARD S. RUBIN 509
The Structural Stability of Tobacco Mosaic Virus. *By* D. L. D. CASPAR 519
The Structure of Small "Spherical" Viruses. *By* BEATRICE S. MAGDOFF 522
French Guiana: Myths and Realities. *By* DAVID LOWENTHAL 528
Psychological Self-Consistency. *By* HERMAN A. WITKIN 541
Conferences Held:
Biochemistry and Pharmacology of Compounds Derived from Marine Organisms 546
Fundamentals of Psychology: The Psychology of Thinking 550
Biochemical Aspects of Microbial Pathogenicity 552
Late Mesozoic and Cenozoic Events in Natal, South Africa. *By* JACK JOSEPH FRANKEL 565
The Psychoprophylactic Preparation of Pregnant Women for Childbirth in the Soviet Union. *By* THOMAS L. BALL 578
Impressions of Soviet Surgery. *By* D. REES JENSEN 581
Medicine and Psychiatry in the Union of Soviet Socialist Republics. *By* ARNOLD A. HUTSCHNECKER 585
Arab and United States Children: Some Psychological Comparisons. *By* WAYNE DENNIS 589
The Problem of Autonomy and Diversity of Anthropology as a Science. *By* ERNEST HARMS 606
Some Meteorologically Important Relationships Between the Ocean and Atmosphere. *By* G. NEUMANN 615
Effect of Attenuated Tumor Tissue Injections on the Sarcoma 1A Ascites Tumor in the A/Jax Mouse. *By* JONATHAN KAPLAN 634
Apical Dominance. *By* ROBERT KOPELMAN 638
Diurnal Rhythms in *Orconectes Virilis*. *By* CHARLENE SUTIN 643
Behavior of Transplanted Hearts in Platyfish. *By* ROBERT R. WEINSTEIN 647
The Effect of Heredity on Epidermal Cross Grafting in Cactus Plants. *By* SIDNEY M. KENT 653
Present Status of Oncolytic Virus Studies. *By* CHESTER M. SOUTHAM 657
French Contribution to Space Research. *By* E. VASSY 674
Conferences Held:
New Diuretics and Antihypertensive Agents 680
Deuterium Isotope Effects in Chemistry and Biology 684
Human Decisions in Complex Systems 687
Thermodynamics and Mechanics of Polymer Systems 690
Culture, Society, and Health 692

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SER. II, VOL. 22 *November 1959* NO. 1

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OF
THE NEW YORK ACADEMY OF SCIENCES

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CONTENTS

In Memoriam: Cornelius Packard Rhoads. *By* DAVID A. KARNOFSKY 3
Some Stratigraphic Problems of the Cow Head Area in Western Newfoundland. *By* C. H. KINDLE AND H. B. WHITTINGTON 7
Studies on the *in Vitro* Accumulative Process for Sulfate Ions of Mammalian Kidney Cortex. *By* INGRITH J. DEYRUP AND JEANETTE C. RODNAN 19
Energy Production from Nuclear Reactions. *By* HARRY SOODAK 26
The Use of an Analogue Computer for Measurement of Respiratory Depression. *By* J. WELDON BELLVILLE AND JOHN C. SEED 34
Enzymatic Autoinduction and the Hypothesis of Intracellular Permeability Barriers in *Neurospora*. *By* S. R. GROSS 44
Dowry and Inheritance in Modern Greece. *By* ERNESTINE FRIEDL 49
Conferences Held 55
New Members 65

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Transactions of The New York Academy of Sciences, Series II, Volume 22, No. 1, November 1959

Second-class postage paid at New York, N. Y. This publication is distributed to members and is published monthly from November to June, inclusive, by The New York Academy of Sciences, 2 East Sixty-third Street, New York 21, N. Y.

Executive Director: EUNICE THOMAS MINER

CORNELIUS PACKARD RHOADS
(1898-1959)

IN MEMORIAM

Cornelius Packard Rhoads (1898-1959)

Cornelius Packard Rhoads was born June 20, 1898 in Springfield, Mass. and he died unexpectedly of a heart attack at his home in Stonington, Conn., August 13, 1959. His passing marks the end of an era, for which he was largely responsible, an era when laboratory and clinical research on cancer expanded enormously to achieve the stature of a mature scientific discipline.

Rhoads, the son of a physician, dedicated his life to the medical sciences and, in particular, during the past twenty years to cancer research. He attended high school in Springfield, Mass. and received his A.B. degree from Bowdoin College in Brunswick, Me. Following this he studied medicine at Harvard and graduated cum laude in 1924. While he planned to go into surgery, during his surgical internship at Peter Bent Brigham Hospital in Boston he developed pulmonary tuberculosis and spent a year at the Trudeau Sanitorium in Saranac Lake, N. Y. This event diverted him from the apparently more strenuous career of a surgeon to become a pathologist, but in actuality his working day, in time and sustained effort, exceeded that of the vast majority of surgeons. After two years as instructor in pathology at Harvard Medical School, he joined the staff of The Rockefeller Institute. He worked there from 1928 to 1939, serving as an Associate Member from 1933 to 1939, and made a number of important contributions in his studies on immunity in poliomyelitis, on the causes of anemia, and on experimental leukemia. His training and early work, in retrospect, seemed deliberately designed to prepare him for the role he achieved as the leading figure in the broad domain of cancer research. He married Katherine Southwick Bolman in 1936, and she was his constant companion and devoted aide until the time of his death.

In 1939, upon the retirement of James Ewing, one of the greatest of United States pathologists, Rhoads was appointed director of Memorial Hospital in New York, N. Y. In assuming direction of a hospital devoted to the care of patients with cancer, Rhoads immediately undertook to develop a research attack on the disease. World War II intervened, however, and he was called to active service in the United States Army Medical Corps, as chief of the Medical Division of the Chemical Warfare Service with the rank of colonel. He was charged with the responsibility of determining the toxicological effects and military value of various war gases, of developing sensitive methods of gas detection, and of devising practical methods to protect people exposed to these gases. While he spent a full work week at his military duties, Rhoads maintained

his direction of Memorial Hospital during week-end visits to New York. For his military contribution he was awarded the Legion of Merit. Rhoads' military experience had a fundamental influence on his attitude toward scientific research. In the war effort he directed a scientific program oriented toward clear objectives and staffed by scientists of many disciplines who were willing to pool their abilities to solve an urgent problem.

When Rhoads returned full time to his post as director of Memorial Hospital, he mounted a full-scale and many-pronged attack on cancer. With a clear vision of his goals, and with a sense of mission that he readily communicated to others, he was able to interest people in the program, develop financial support, and attract a staff of able investigators to the difficult field of cancer research. Alfred P. Sloan and Charles F. Kettering were deeply impressed by his direct approach to the cancer problem, and they established the Sloan-Kettering Institute for Cancer Research as an integral part of Memorial Center. The city of New York contributed the James Ewing Hospital for the treatment of cancer, and Memorial Center continued to grow with the increase in its responsibilities and the intensification of its research program. The Sloan-Kettering Division was established as a graduate school of Cornell University. In 1959 the Walker Laboratory in Rye, N. Y., a unit of the Sloan-Kettering Institute, was opened, and other construction plans were initiated to make Memorial Center and the Sloan-Kettering Institute the greatest center for cancer research in the world. Since 1953, as Scientific Director of the Sloan-Kettering Institute, Rhoads dominated the scene and directed the development of his institution with a single-minded purpose.

Meanwhile, due in large part to his example and effort, cancer research was intensified throughout the United States and the world. He was largely responsible for organizing the Committee on Atypical Growth and later the Committee on Growth of the National Research Council, which wisely, for a period of ten years, allocated the research funds of the American Cancer Society to stimulate interest in cancer in many universities and among different scientific disciplines. He was active in the American Cancer Society during the period of its rapid growth following the end of World War II. The cancer chemotherapy program of the Sloan-Kettering Institute became the model for the Cancer Chemotherapy National Service Center of the National Institutes of Health, which is financing and directing a world-wide search for a chemical treatment of cancer.

Rhoads was a member of many professional societies, and filled many responsible roles on governmental and medical society committees. He was Councilor of The New York Academy of Sciences from 1955 to 1957, and had been Vice-President since 1957.

He was the recipient of many honors. He was given Doctor of Science degrees by Bowdoin College in 1945 and Williams College in 1942, and

an honorary LL.D. by the University of Saskatchewan in 1958. He received the New York City Cancer Committee's Clement Cleveland Award in 1948, the American Cancer Society's Annual National Award in 1955, the Royal College of Surgeons' Walker Prize in 1956, and the French Legion of Honor in 1959.

In the last twenty years of his life the conquest of cancer was Rhoads' overriding ambition. Rhoads established groups and actively participated in programs on steroid chemistry, nucleic acid metabolism, epidemiology of cancer, carcinogenesis, cancer chemotherapy, genetics, pathology, and the treatment of cancer. He took young and in many cases unseasoned investigators and provided them with facilities, encouragement, and direction His confidence was rarely misplaced, and many of these men developed into productive and distinguished scientists. Despite his dedication to the control of cancer, his staff was permitted complete freedom to develop its own research programs, even though they sometimes veered from the direct attack on cancer.

Rhoads was a warm and considerate person, always available to those who wished to discuss their problems. His many personal favors, often given without request because he anticipated a need, were conferred on many people, and he was a source of information and comfort to thousands of patients and their families who called on him for advice. It was perhaps because of his contact with patients that he felt strongly that scientists must have a sense of obligation to humanity and must organize direct attacks on problems of social importance, rather than hope that practical knowledge will appear as a by-product of work stimulated only by the desire to know or by curiosity. This is not to minimize his interest in basic research, for he would defend the cause of free research everywhere. Rhoads had highly developed critical faculties that enabled him to see to the heart of a research proposal, yet he exhibited what appeared to be an almost excessively open mind, giving every proposal a fair hearing and attempting to extract a germ of truth from it. He became the focal point of leadership in all aspects of cancer research; scientists throughout the United States and throughout the world came to him for help and advice, and none left without a feeling of direction and inspiration. In meetings and conferences, Rhoads gave the proceedings a sense of dignity and purpose, a reflection of his dedicated leadership.

One of his greatest attributes was his boundless optimism and hope. Rhoads knew that cancer would ultimately be a curable disease, and no place was too obscure to search. No one could feel discouragement in his presence, and the sense of ultimate victory that he radiated, although it was not a scientific judgment, was of the utmost value. It is clear that nothing of value can be accomplished unless a man feels it is possible and is able to communicate this feeling to others.

The buildings that he built will endure, the great public support that he stimulated for cancer research will continue, and the programs that he

helped to found will continue to expand in scope and productivity. What will be missed more than anything else—and this is the major tragedy of his premature death—is his sense of purpose and direction, his emphasis on the dignity of the search, and his assumption of the responsibility of solving a problem and defeating a disease that causes such pain and distress.

Prepared by
David A. Karnofsky
October, 1959

SECTION OF GEOLOGICAL SCIENCES

SOME STRATIGRAPHIC PROBLEMS OF THE COW HEAD AREA IN WESTERN NEWFOUNDLAND*

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As a result of field work undertaken in 1955 Kindle and Whittington published a paper on "Stratigraphy of the Cow Head Region, Western Newfoundland" in the March 1958 number of the *Bulletin of the Geological Society of America*. Reference is made to this earlier paper for an introduction to the present paper, which details the results of some of the work done since that time. The location of places referred to in this paper is shown in FIGURE 1.

In the field seasons of 1957 and 1958 our collections of fossiliferous boulders from the Cow Head group of conglomerates were considerably enlarged from both Cambrian and Ordovician strata. During the summer of 1959 the Cambrian boulders were unpacked and their faunas partially identified. Each boulder has been numbered so that a record of the associations of the fossils will be preserved by their numbers. Three hundred and sixty-one of these Cambrian fossiliferous boulders are now spread out in the drawers of cabinets at City College.

These Cambrian faunas range from the *Bathyuriscus-Elrathina* zone of the middle Cambrian to the top of the Cambrian. Many new species are represented that are not mentioned in this paper. We shall select a few boulders from the sequence of strata at five localities to see, first, what is the sequence of faunas at each of these localities and, second, how the sequence at one locality compares with that at other localities.

In our earlier paper we showed the Cow Head group to consist of a series of conglomerates interspersed with normal strata of limestone and shale. The unusual size of many of the boulders might be due to submarine sliding with accompanying turbidity current as the transporting medium. We noted that the fossils in any one conglomerate were commonly the same age as those in the normal sediments immediately underlying it. This regularity in successive ages of the fossils in boulders from succeeding conglomerates is a striking feature of the Cow Head group. One would expect some exceptions to this regularity in these variable con-

*This paper, illustrated with slides, was presented at a meeting of the Section on October 5, 1959.

The Section of Chemical Sciences held a meeting on October 6, 1959, at which a paper entitled "Molecular Mechanisms of Flow" was presented by Melvin Mooney of Mountain Lakes, N. J. No copy of this paper has been received.

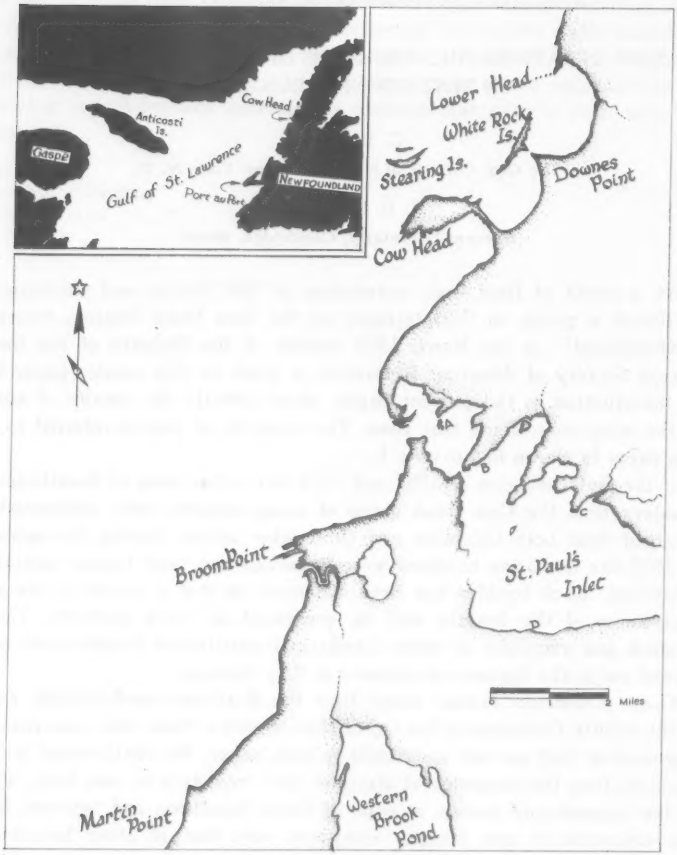

FIGURE 1. Location of the Cow Head area.

glomerates, and our work has shown that some exceptions do occur, as we shall note further on.

The Cambrian Sequence at Cow Head

If one refers to the geological map of Cow Head Peninsula as given in our earlier paper, the beds marked 1, 2, 3, and 4 are those where the *Cedaria-Crepicephalus* fauna was found. We now know this faunal zone is also found in the first twenty feet of conglomerate along the sea edge extending southwest to a point in front of the cliff edge marked Crow's Nest. We are thus enabled to list the following sequence of faunas

beginning at the sea edge and going up toward the high cliff from in front of and slightly west of the lighthouse on Cow Head (TABLE 1).

TABLE 1

SEQUENCES OF CAMBRIAN FAUNAS

Cow Head		Hickey Cove (Three-fourths and 1 mile east of Broom Point)	
77	*Keithia* *Keithiella* *Plethometopus* *Rasettia*	107	*Bellaspis* *Keithia* *?Loganopeltis* *Loganopeltoides* *Onchonotus* *Stenopilus*
75	*Keithiella* *Loganellus* *Loganopeltoides* *Theodenisia*	148	*Keithiella* *Onchonotus* *Plethometopus* *Phoreotropis* *Richardsonella* *Theodenisia*
72	*Loganellus* *?Olenid pygidium*		
58	*?Loganellus* *?Parabolinoides* *?Orygmaspis*	102	*Calymenidius* *Lecanopyge* *Loganellus* *Phoreotropis* *Theodenisia*
56	*Glyptagnostus reticulatus* *Proaulacopleura*	104	*Bienvillia*
54	*Cheilocephalus* *Dunderbergia* *Dytremacephalus*	119	*Buttsia* *Irvingella*
48	*Blountia* *Aphelaspis*	129	*Dytremacephalus*
		125	*Dunderbergia*
38	*Cedaria* *Coosella* *Crepicephalus* *Deiracephalus*	130	*Blountia* *Aphelaspis*
		138	*Cedaria* *Crepicephalus* *Tricrepicephalus*
		127	*Meteoraspis*

Boulder 38, twenty feet above the sea, has *Cedaria* and *Crepicephalus*; boulder 48, twenty-seven feet above the sea, has *Aphelaspis* and *Blountia*; boulder 54, thirty feet above the sea, has *Cheilocephalus*, *Dunderbergia*, and *Dytremacephalus* (FIGURE 3*g*); boulder 56, forty feet above the sea, has *Glyptagnostus reticulatus* (FIGURE 3*f*) and *Proaulacopleura*; boulder 58, two thirds of the distance from the lighthouse west to the fault nick, has ?*Loganellus*, ?*Parabolinoides*, and ?*Orygmaspis*; boulder 72, in the succeeding conglomerate, has *Loganellus* and a ?*Olenid pygidium*; boulder 75, thirty feet below the upper sandstone rib or sixty feet below *Dic-*

TABLE 2

SEQUENCES OF CAMBRIAN FAUNAS

One-half and 2/3 mile east of Broom Point		Below *Dictyonema* beds east of Broom Point	
176	?*Bienvillia*	247	?*Symphysurina*
208	?*Parabolinella* *Litagnostus*	245	*Keithia* *Idiomesus* *Plethopeltis*
172	*Loganopeltoides* *Phoreotropis* *Taenicephalina*	240	?*Bellaspis* *Idiomesus* *Keithia* *Leiocoryphe* *Loganopeltoides* *Onchonotus* ?*Plethometopus* *Paranorwoodia* *Theodenisia*
178	*Irvingella*		
168	*Irvingella (?Parairvingella)* *Oligometopus*		
202	*Cheilocephalus* *Dunderbergia* *Dytremacephalus* *Glaphyraspis* *Labiostria* *Pterocephalops*	239	*Bayfieldia* *Onchonotus*
166	*Dunderbergia* *Onchopeltis* *Pterocephalops*	231	*Bayfieldia* *Idiomesus* *Keithia* *Leiocoryphe* ?*Plethometopus* *Stenopilus* *Westonaspis*
200	*Blountia* *Aphelaspis*		
199	*Meteoraspis*		
198	*Crepicephalus*		

TABLE 3

SEQUENCES OF CAMBRIAN FAUNAS

North of Sandy Cove, Broom Point		South of Sandy Cove, Broom Point	
295	*?Hystricurus* *?Neoagnostus* *Symphysurina* *Ulrichaspis*	488	*"Acidaspis" ulrichi* *Idiomesus* *Leiocoryphe* *Plethometopus* *Richardsonella* *Stenopilus*
291	*Leiocoryphe* *Plethopeltis* *Theodenisia*		
277	*Onchopeltis* *Pterocephalops*	480	*Cheilocephalus* *?Drumaspis*
		468	*Phalacroma* cf. *glandiforme*
272	*Agraulos* *Kootenia* *Peronopsis* cf. *fallax* *?Solenopleura* *Solenopleurella*	467	*?Blountiella* *?Brachyaspidion* *?Brassicicephalus* *Catillicephala* *Komagnostus* *?Matania* *?Menomonia* *Onchonotopsis*
268	*Bathyuriscus* cf. *adaeus* *Pagetia* *Peronopsis*	458	*Phalacroma* *Ptychagnostus* cf. *aculeatus*
		452	*Eodiscus scanicus* *Parasolenopleura* *?Poliella* *?Solenopleura* *Tomagnostus* *Ptychagnostus atavus*
		425	*Centropleura*
		413	*Agraulos* *Kootenia* *Solenopleura* *Zacanthoides*
		410	*?Orriella*
		372	*?Acrocephalops* *Pagetia*
		357	*Bathyuriscus* *Ogygopsis* *Olenoides*
		356	*Bathyuriscus* cf. *adaeus* *?Solenopleura* *Zacanthoides*

tyonema, has *Keithiella*, *Loganellus*, *Loganopeltoides*, and *Theodenisia*; finally, boulder 77, just below the upper sandstone rib or thirty feet below *Dictyonema*, has *Keithia*, *Keithiella* (FIGURE 3a), *Plethometopus*, and *Rasettia*.

As may be seen by reference to TABLE 4, all these boulders from the Cow Head sequence fit the standard sequence of Cambrian faunas. However, we should mention that boulder 40, collected thirty feet above the sea, with *Hypagnostus parvifrons*, *H.* cf. *truncatus*, *Peronopsis* cf. *scutalis*, *Ptychagnostus* cf. *atavus*, and *Triplagnostus* cf. *hybridus*, does not fit in this sequence. It appears to be older than any other fauna found on Cow Head.

The Sequence at Hickey Cove

If we refer to FIGURE 2 we see that the boulders from the east side of Hickey Cove are numbered from 100 to 107; those from the west side of the Cove, from 116 to 148. The numbers progress from the lowest strata at the sea edge with smaller numbers to higher strata inland with higher numbers. If we now refer to TABLE 1, we see the faunas arranged according to their geological age. We note this sequence is not in the order in which they were found on the west side of Hickey Cove. Boulder 119, fifteen feet above the sea, with *Irvingella* (FIGURE 3e), and *Buttsia*, was found nearer the base than boulder 138, forty feet above the sea, with *Cedaria*, *Crepicephalus* and *Tricrepicephalus*. Even closer to the sea is boulder 118 with a post - *Elvinia* or *Conaspis* zone faunule. At this same level of ten feet above the sea is boulder 117 with *Plethopeltis*, *?Keithiells*, and *?Resseraspis*. Thus, it may be seen that along the west side of Hickey Cove boulders ranging in age from the *Cedaria-Crepicephalus* zone to the *Hungaia magnifica* zone are mixed.

Sequence One-Half and Two-Thirds Mile East of Broom Point

Boulders at two-thirds mile east of Broom Point are numbered from 160 to 181; those from one-half mile east of Broom Point are numbered from 198 to 214 (FIGURE 2). Making allowance for these two sets of numbers on the same column in TABLE 2, it is noted that the mix-up exhibited on the west side of Hickey Cove does not occur here (boulder 176 was collected about ten feet above 178 and boulder 172 at the same level as boulder 178, a laboratory error in numbering). The oldest faunas are found in the lowest conglomerate layers, intermediate faunas are higher, and the youngest are in the highest layers exposed.

At the base are boulders of the *Cedaria-Crepicephalus* zone, followed by *Aphelaspis*. Above this the late *Aphelaspis-Dunderbergia* zone is represented by *Glaphyraspis*, *Cheilocephalus*, *Labiostria*, *Dunderbergia* (FIGURE 3*h*), *Dytremacephalus*, *Onchopeltis*, and *Pterocephalops*. Higher layers with *Irvingella* and *Oligometopus* represent the *Elvinia* zone. The

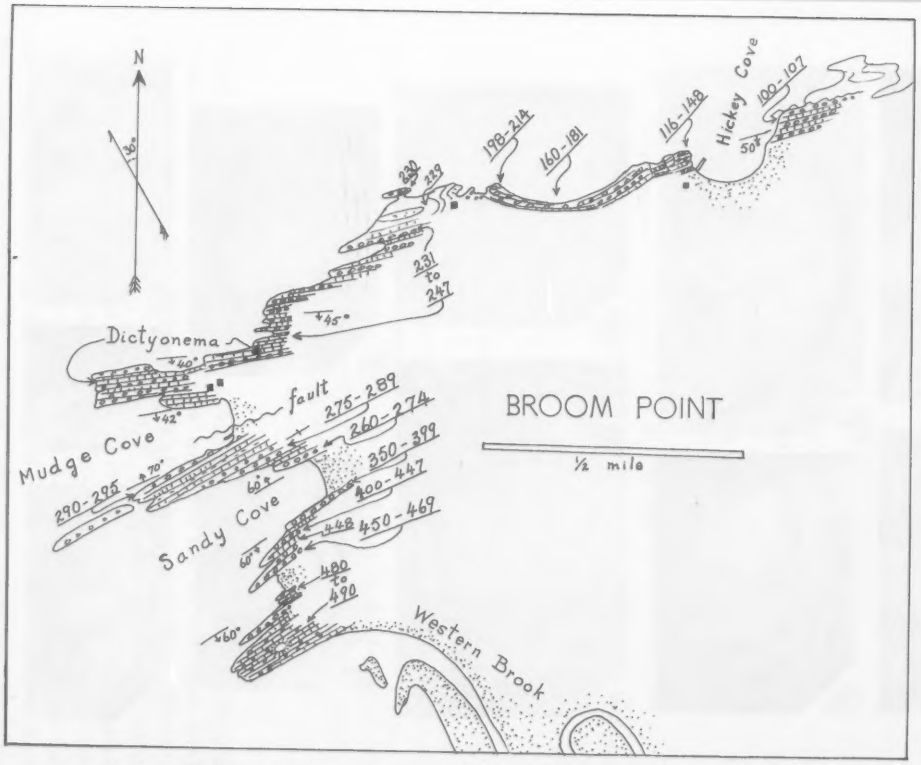

FIGURE 2. Broom Point, showing where the numbered fossiliferous boulders were collected.

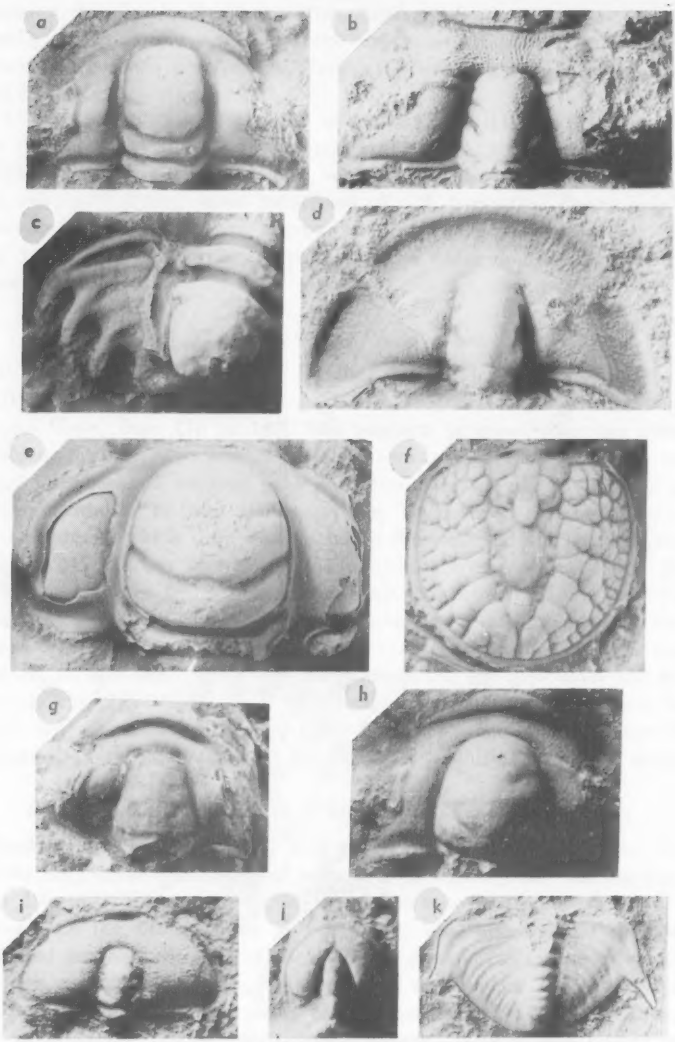

FIGURE 3 (*a*) *Keithiella* from boulder 74. X5. (*b*) *Ulrichaspis* from boulder 295. X2. (*c*) *Bayfieldia* from boulder 232. X4. (*d*) *Loganopeltoides* from boulder 172. X3. (*e*) *Irvingella* from boulder 119. X5. (*f*) *Glyptagnostus* from boulder 56. X8. (*g*) *Dytremacephalus* from boulder 54. X5. (*h*) *Dunderbergia* from boulder 201. X5. (*i*) *Menevia* cf. *venulosa* from boulder 450. X7. (*j*) *Eodiscus scanicus* from boulder 452. X6. (*k*) *Bathyuriscus* cf. *adaeus* from boulder 268. X6.

TABLE 4

Standard sequence of some Cambrian faunal zones	Fossiliferous boulders of similar ages
Saukia *Prosaukia-Ptychaspis*	75, 77, 107, 148, 240, 245, 291, 295, 488, 549 Martin Pt.
Conaspis	58, ?102, ?104, ?172, ?176, 208.
Elvinia	124, 168, 178, ?480.
Late *Aphelaspis* *Dundergergia*	54, ?56, 125, 129, 166, 202, 277, 528 Martin Pt.
Aphelaspis	48, 130, 200.
Crepicephalus *Cedaria*	38, 127, 138, 198, 199.
Bolaspidella	? 458, 467, 468.
Poliella-Paraehmania	? 452, 640 Stearing Island
Olenoides-Ortia-Marjumia	? 410, 413, 425 ? 606, 618 White Rock Island
Bathyuriscus-Elrathina	356, 357, 372, 268, 272.

Conaspis zone is represented by boulders 208 with ?*Parabolinella* and *Litagnostus*; 211 with *Conaspis* zone trilobites and at the same level 212 (the "brachiopod boulder") with *Pseudagnostus* cf. *cyclopyge*, *Levisella*, *Loganopeltoides* (FIGURE 3*d*), and *Taenicephalina*. Boulder 176 with *Bienvillia* may also belong in this zone. If so, the *Hungaia magnifica* zone is not represented here, but is exposed further to the east. The beds here exhibit an anticlinal structure plunging down to the east with some confusing convolutions. We now consider boulders from the younger beds beyond the confused structure separating these two sequences.

Sequence Below The Dictyonema Beds East of Broom Point

Boulder 230 has *Bayfieldia* (FIGURE 3c), ?*Loganopeltoides*, ?*Onchonotopsis*, and *Theodenisia*. Boulder 231, from the eleventh conglomerate

below the *Dictyonema* beds, has *Bayfieldia, Idiomesus, Keithia, Leiocoryphe, ?Plethometopus, Stenopilus,* and *Westonaspis.* Boulder 239, from the ninth conglomerate below *Dictyonema,* has *Bayfieldia* and *Onchonotus.* Boulder 240, from the sixth conglomerate below *Dictyonema,* has *?Bellaspis, Idiomesus, Keithia, Leiocoryphe, Loganopeltoides, Onchonotus, ?Plethometopus, Paranorwoodia,* and *Theodenisia.* From the fifth conglomerate (fourth and fifth) below *Dictyonema,* boulder 245 gives us *Keithia, Idiomesus,* and *Plethopeltis.* From the first conglomerate below *Dictyonema,* boulder 247 gives us *?Symphysurina.* All these boulders seem to have a rather similar fauna and, as may be seen in TABLE 2, the sequence of the right-hand column seems to belong above the sequence in the left-hand column. This *Hungaia magnifica* zone may be equivalent to both the *Saukia* and the *Prosaukia-Ptychaspis* zones of the mid-continent. Boulder 247 is interesting, as *Symphysurina* is commonly considered an Ordovician trilobite. We meet this genus again in our next sequence, that from Sandy Cove to Mudge Cove.

Sequence North of Sandy Cove, Broom Point

Sandy Cove is in our notebooks as the anticlinal cove at Broom Point. As befits an anticline, the oldest conglomerate corresponds well in its fauna with that of the oldest conglomerate south of the cove. Higher conglomerates, on the other hand, do not correlate so readily. Reference is made to FIGURE 2 for location of boulders and to TABLE 3 for sequence of faunas.

Boulder 268 with *Bathyuriscus* cf. *adaeus* (FIGURE 3*k*), *Pagetia,* and *Peronopsis* and boulder 272 with *Agraulos, Kootenia, Peronopsis* cf. *fallax, ?Solenopleura,* and *Solenopleurella* represent the *Bathyuriscus-Elrathina* faunal zone. The next thick conglomerate above this is represented by boulder 277 with *Onchopeltis* and *Pterocephalops* of the late *Aphelaspis-Dunderbergia* zone. Fossils were met with again only in the highest conglomerate, on the south side of Mudge Cove. Here boulder 291 yielded *Leiocoryphe, Plethopeltis,* and *Theodenisia*; boulder 295 yielded *?Hystricurus, ?Neoagnostus, Symphysurina,* and *Ulrichaspis* (FIGURE 3*b*). Boulder 291 and several others from the same conglomerate have the usual late Cambrian trilobites. Boulder 295, however, has puzzling aspects. *Ulrichaspis* was described from a boulder with no associations; therefore, it gives us no clue to the age. *Symphysurina* is commonly regarded as Ordovician, but in boulder 247 what appears to be this genus is found below *Dictyonema.*

Cambrian Sequence South of Sandy Cove, Broom Point

The oldest conglomerate south of Sandy Cove has supplied boulder 356 with *Bathyuriscus* cf. *adaeus, ?Solenopleura, Zacanthoides;* boulder 357 with *Bathyuriscus, Ogygopsis* and *Olenoides*; also boulder 372 with

?*Acrocephalops* and *Pagetia*. These represent the *Bathyuriscus-Elrathina* faunal zone.

The five-foot conglomerate above this has produced boulder 410 with ?*Orriella*; boulder 413 with *Agraulos, Kootenia, Solenopleura,* and *Zacanthoides;* also boulder 425 with *Centropleura*. This may represent the *Olenoides-Orria-Marjumia* faunal zone.

The bedded limestone above this conglomerate has yielded *Meneviella* cf. *venulosa* (FIGURE 3*i*) and *Tomagnostus* cf. *fissus*. This collection has been assigned the number 448.

About ten feet above this the "50-foot" conglomerate begins. The same fauna as 448 is found there in boulder 450 and of similar age in boulder 452 with *Eodiscus scanicus* (FIGURE 3*j*), *Parasolenopleura*, ?*Poliella*, ?*Solenopleura, Tomagnostus,* and *Ptychagnostus atavus*. Of the standard faunal zones on TABLE 4 these may represent the *Poliella-Paraehmania* zone.

The 50-foot conglomerate has also produced boulders with a younger fauna than these, but these younger fauna are probably older than the *Cedaria-Crepicephalus* fauna of Cow Head. This includes boulder 458 with *Phalacroma* and *Ptychagnostus* cf. *aculeatus*; boulder 468 with *Phalacroma* cf. *glandiforme;* and also boulder 467 with ?*Blountiella*, ?*Brachyaspidion*, ?*Brassicicephalus, Catillicephala, Kormagnostus*, ?*Matania*, ?*Menomonia*, and *Onchonotopsis*.

Above the 50-foot conglomerate is an unexposed interval of fifty yards. In the first conglomerate above this interval, boulder 480 has *Cheilocephalus* and ?*Drumaspis*, possibly of the age of the *Elvinia* zone.

The higher conglomerates yield fossiliferous boulders of late Cambrian age such as boulder 488, which has *"Acidaspis" ulrichi, Idiomesus, Leiocoryphe, Plethometopus, Richardsonella*, and *Stenopilus*.

In this sequence of Cambrian faunas we note an orderly arrangement from the oldest to the youngest (TABLE 3). Not all the faunal zones are present, however. Notably absent is the *Cedaria-Crepicephalus* fauna that occupied a great thickness of conglomerates on Cow Head. Also missing are the *Aphelaspis,* late *Aphelaspis-Dunderbergia*, and *Conaspis* faunas. These last were well represented one mile away, east of Broom Point. This reminds us that these conglomerates thicken and thin rapidly in a short distance. It suggests, too, that conglomerate outcrops elsewhere, even a short distance away, may provide considerable additions to the faunas now known.

Cormorant Head

At this point a question arises concerning the exposures at Cormorant Head on the Port-au-Port Peninsula. Are the conglomerates there related in origin to faults exposed in the cliffs, or are they of similar origin to those already discussed?

We believe they are of the same origin. A brief trip was made to Cormorant Head late in the 1958 field season. The exposure there as seen from the sea at the junction of the thick conglomerate and the carbonate rock is not as clear as could be desired. The carbonates here display considerable disturbance. They bend down and are cut by several faults near the contact with the conglomerate. The picture clears as one follows the cliffs to the southeast. The carbonate rocks descend gently in this direction, with the dip increasing as one proceeds. About two miles along the shore it is 45°, and the rocks are overlain by a series of conglomerates dipping at the same angle. These seem to be the same conglomerates as those that occur north of Cormorant Head. The area between the conglomerate outcrops must be in the nature of an asymmetric anticline complicated by faulting at the northwest side. This faulting must have accompanied the folding long after the conglomerates were deposited.

Acknowledgments

We are indebted to the National Science Foundation, Washington, D.C., for Research Grant G-4189, which has enabled us to continue our work in this area; and to Gunnar Henningsmoen we are grateful for two weeks' assistance in fossil collecting, and to A. R. Palmer for suggestions in the identification of some specimens.

STUDIES ON THE *IN VITRO* ACCUMULATIVE PROCESS FOR SULFATE IONS OF MAMMALIAN KIDNEY CORTEX*

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The mechanisms underlying accumulation of S^{35}-labeled sulfate ions by renal tissue *in vitro* are still far from clear. This phenomenon, first described several years ago (Deyrup and Ussing, 1955), continues to be of interest because it offers a possible approach to the analysis of normal renal reabsorption of sulfate ions, and/or more general problems of kidney sulfate metabolism. The experiments described in this paper were undertaken to establish the effects of cations of the alkali metal series on sulfate accumulation. Previously it had been shown that much more radiosulfate is taken up in a given period of time from solutions containing K^+ than from media containing Na^+ ions (Deyrup and Ussing, 1955), and that the specific activity of sulfate was significantly lower in the presence of Na^+ than when K^+ was the principal cation of the incubation medium (Berglund and Deyrup, 1956).

Methods

In general, experiments were performed along lines established by the classic work of Forster and his co-workers (1950, 1956), Cross and Taggart (1950), and others in studying *in vitro* renal accumulative processes for dyes, *p*-amino hippurate, and other compounds. Thin (0.3 to 0.45 mm.) slices of kidney cortex were prepared from kidneys of rats, and these slices were incubated, under conditions controlled with respect to ambient medium composition, temperature, atmosphere, and shaking rate, in the presence of tracer concentrations of $S^{35}O_4^=$ with $S^{32}O_4^=$ (usually the total sulfate concentration was 0.1 μM). The incubation media used were isosmotic (300 mOs M/l.) solutions prepared by mixing 0.3 M sucrose with 0.15 M solutions of LiCl, NaCl, KCl, RbCl, or CsCl. The final concentration of the alkali metal cations was 0.04 M.

After incubation for 30 to 60 min., the kidney tissue was removed from the incubation medium and divided into 2 portions for determination of water content (wet and dry weight measurements) and of S^{35} content (helium-isobutane flow counter), respectively. The S^{35} content of each medium was measured, and a comparison was made between the virtual

* This paper, illustrated with slides, was presented at a meeting of the Section on October 12, 1959. The work described in this article was supported by Research Grant H-2061 from the National Heart Institute, Public Health Service, Bethesda, Md.

S^{35} concentration of the tissue ($T = \frac{\text{tissue } S^{35} \text{ content}}{\text{tissue water content}}$) and the S^{35} concentration of the incubation medium (M). Thus, a virtual gradient = T/M was calculated for every tissue sample. A gradient of 1 would indicate that the virtual concentration in the tissue was identical with that of the medium, as might be expected if sulfate entered the tissue cells by simple diffusion. In fact, however, gradients greater than 1 were always seen under the conditions of the experiments. Such high S^{35} gradients, which are correlated with an absolute increase in tissue sulfate content (Berglund and Deyrup, 1956) and may be abolished by metabolic inhibitors (Deyrup, 1956), are interpreted as evidence for renal accumulation of sulfate *in vitro*.

Results

Renal tissue slices are capable of accumulating radiosulfate from media containing the anion in tracer amounts, both at low temperature (0° C.) and at the higher temperature range commonly used in *in vitro* metabolic experiments (25 to 38° C.). At 0° C., relatively low virtual concentration gradients between slice and medium (T/M = 3.5 to 4.0) are achieved over the course of 30 min. in incubation media consisting primarily of KCl and sucrose, whereas much higher gradients (T/M = 11 to 15) are attained if the ambient temperature ranges from 25 to 38° C. (Deyrup *et al.*, 1959). FIGURE 1 summarizes results from a series of experiments in which the S^{35} accumulative process was compared in media containing sucrose and LiCl, NaCl, KCl, RbCl, or CsCl. It may be noted that the results are strikingly dependent upon temperature. At 0° C. the highest T/M gradients occurred in tissues in solutions containing LiCl or NaCl, whereas the lowest gradients occurred in these media at 38° C. The T/M gradients developed in solutions of RbCl did not differ significantly from those in KCl, but slightly lower gradients were seen in the case of CsCl.

When tissues were allowed to develop high S^{35} gradients at 38° C. in the presence of K^+ and were then transferred to media consisting of isosmotic mixtures of sucrose and NaCl or LiCl solutions, the S^{35} was lost rapidly (FIGURE 2). Incubation in NaCl- or LiCl-sucrose solutions did not, however, prevent the subsequent development of high T/M gradients in slices transferred from such solutions to KCl-sucrose. These results indicate a high degree of reversibility of the cation effects.

Phlorizin (4.5×10^{-3} M), known to enhance S^{35} uptake at 38° C. in the presence of Na^+ and K^+ ions, has a similar effect in the presence of Li^+, Rb^+, and Cs^+. Moreover, the characteristic depression of S^{35} T/M gradients with phlorizin seen at 0° C. occurred in the presence of all of the cations tested. These findings are summarized in TABLE 1.

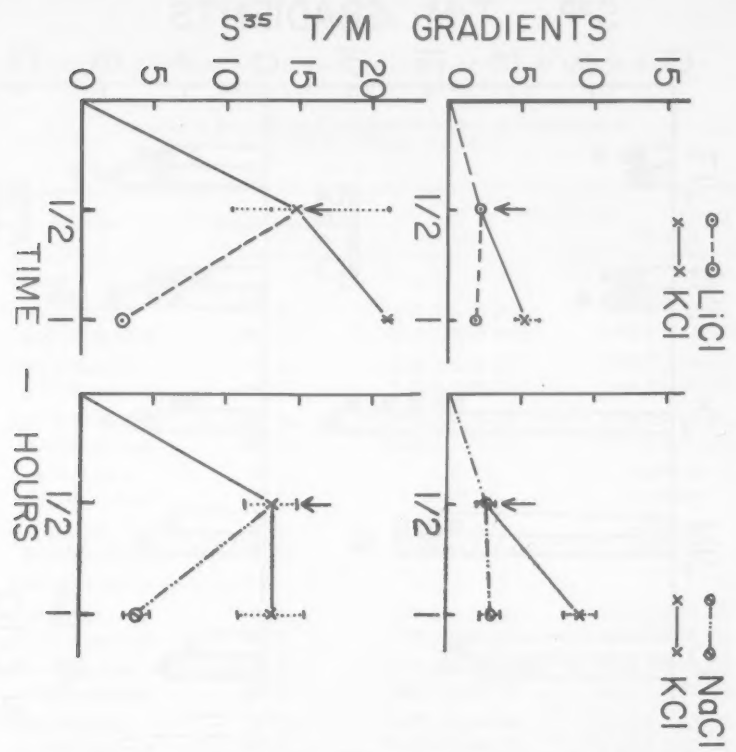

FIGURE 1. Virtual tissue/medium S^{35} gradients developed by kidney cortex incubated for ½ and 1 hour at 0° C. and 38° C. in the presence of 0.22 M sucrose and 0.04 M concentrations of LiCl, NaCl, KCl, RbCl, or CsCl. The columns represent means obtained from the results of the individual experimental values, shown by circles.

Discussion

The observed effects of the cations tested on net sulfate uptake are not surprising in one sense, for the pattern shown in these experiments is revealed again and again in the case of physiological processes that seem, in the present state of our knowledge, to be entirely unrelated to one another or to the accumulative mechanisms under study here. Thus, given a process *P* enhanced by K^+, in all probability Rb^+ will have a similar or identical effect, whereas Li^+ and Na^+ will have an opposite effect, or none at all. The action of Cs^+ is somewhat less predictable, although its action tends to parallel that of K^+ and Rb^+, rather than the effects of Li^+ and Na^+. This pattern of effects may be seen in the case of partially isolated enzyme systems (Happold and

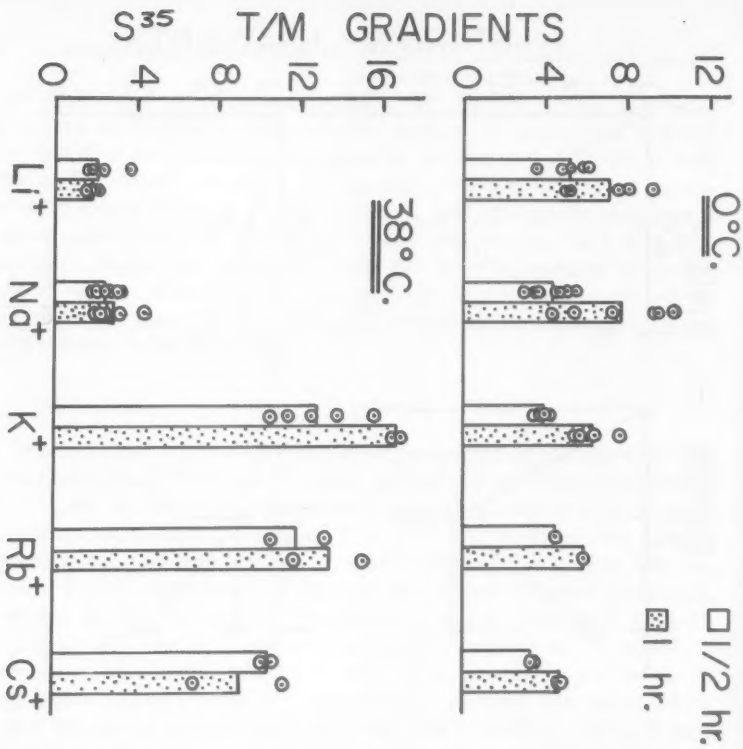

FIGURE 2. Virtual tissue/medium S^{35} gradients developed by kidney cortex incubated in the presence of 0.22 M sucrose and 0.04 M concentrations of LiCl, NaCl, or KCl. At the arrow, portions of the tissues were transferred to media containing different cations (that is, from LiCl to KCl; from KCl to LiCl; from NaCl to KCl; from KCl to NaCl). Control tissues were allowed to remain in the original incubation medium. The points represent means with range of individual values (LiCl/KCl series) or means with standard deviations (NaCl/KCl series). All incubations were carried out at 38° C. with oxygen atmosphere.

Struyvenberg, 1954; von Korff, 1953), and in many simple or complex processes of bacteria (MacLeod and Snell, 1948) and higher plants and animals (Sexton and Meyer, 1955; Ussing and Zerahn, 1954; Huf and Wills, 1951). No fully satisfactory explanation of this phenomenon has yet been made. Some inferences may be drawn, however, in the case of the renal accumulative mechanism under consideration, as to possible sites of action of the cations in the system. Thus, it may be inferred that the cations probably do not affect the uptake process primarily by a direct action on cell permeability, involving restriction or facilitation of access of the $S^{35}O_4^{=}$ to the intracellular accumulative site. The relatively high gradients achieved by slices incubated at 0° C. in the

TABLE 1

VIRTUAL S^{35} TISSUE/MEDIUM GRADIENTS AFTER 30 AND 60 MIN. OF INCUBATION AT 0° C. AND 38° C.

Figures in Parentheses Indicate Numbers of Experiments on Which Each Mean is Based

Medium	*Temperature*	*Incubation for* 30 min.	60 min.
Control			
0.04 M LiCl + 0.22 M sucrose	0° C. 38° C.	5.12 ± 1.05 (5) 2.17 ± 0.56 (11)	7.05 ± 1.70 (5) 1.82 ± 0.21 (11)
0.04 M NaCl + 0.22 M sucrose	0° C. 38° C.	4.31 ± 0.91 (7) 2.47 ± 0.48 (10)	7.70 ± 2.34 (6) 2.89 ± 0.83 (7)
0.04 M KCl + 0.22 M sucrose	0° C. 38° C.	3.88 ± 0.30 (4) 13.10 ± 1.87 (5)	6.36 ± 0.96 (4) 13.16 ± 2.30 (5)
0.04 M RbCl + 0.22 M sucrose	0° C. 38° C.	4.49 (1) 11.90 (2)	5.84 (1) 13.44 (2)
0.04 M CsCl + 0.22 M sucrose	0° C. 38° C.	3.37 (2) 10.42 (2)	4.73 (2) 9.05 (2)
With phlorizin — 4.5×10^{-3} M			
0.04 M LiCl + 0.22 M sucrose	0° C. 38° C.	1.68 ± 0.13 (5) 2.70 ± 0.30 (6)	3.19 ± 0.65 (5) 3.43 ± 0.65 (6)
0.04 M NaCl + 0.22 M sucrose	0° C. 38° C.	2.97 ± 0.67 (6) 3.82 ± 0.82 (9)	4.74 ± 0.83 (6) 4.83 ± 0.52 (6)
0.04 M KCl + 0.22 M sucrose	0° C. 38° C.	1.67 ± 0.23 (4) 17.55 ± 1.40 (4)	2.45 ± 0.43 (4) 20.65 ± 2.62 (4)
0.04 M RbCl + 0.22 M sucrose	0° C. 38° C.	1.37 (1) 14.88 (2)	2.40 (1) 21.02 (2)
0.04 M CsCl + 0.22 M sucrose	0° C. 38° C.	1.48 (2) 12.08 (2)	2.96 (2) 15.80 (2)

presence of LiCl and NaCl and the rapid loss of $S^{35}O_4^=$ accumulated in KCl-sucrose solutions after transfer to Na^+- or Li^+-containing media argue against a simple effect on cell membrane permeability. Moreover, whatever may be the mechanism accounting for the action of Li^+ and Na^+ on *T/M* gradients for S^{35}, the action is almost completely reversible in the case of Na^+ and partially reversible with Li^+. If an "accumulative site" for sulfate is postulated, in analogy, for instance, to the intracellular sites postulated by Beyer (1950) for phenol red and by Foulks and Miller (1959) for *p*-amino hippurate, then it must be concluded that this site is not irreversibly inactivated in the presence of Li^+ and Na^+, even though only low *T/M* gradients are developed.

Phlorizin, a compound used frequently in physiological experimentation as a specific inhibitor of glucose transport, was shown previously

to have a dual effect on S^{35} accumulation in the presence of K^+ ions, enhancing the net uptake at 0° C. and depressing it at 38° C. (Deyrup *et al.*, 1959). Tentatively, this may be interpreted as evidence that two distinct limiting processes, differentially affected by phlorizin, are involved in sulfate uptake under the two contrasting temperature conditions. Since the 0° C. inhibition and 38° C. enhancement occur regardless of the cationic species present, it seems probable that the phlorizin effects are independent of the latter. Thus, sequential steps in the S^{35} accumulative process may be postulated on the basis of the cation and phlorizin studies. FIGURE 3 presents one of a number of possible

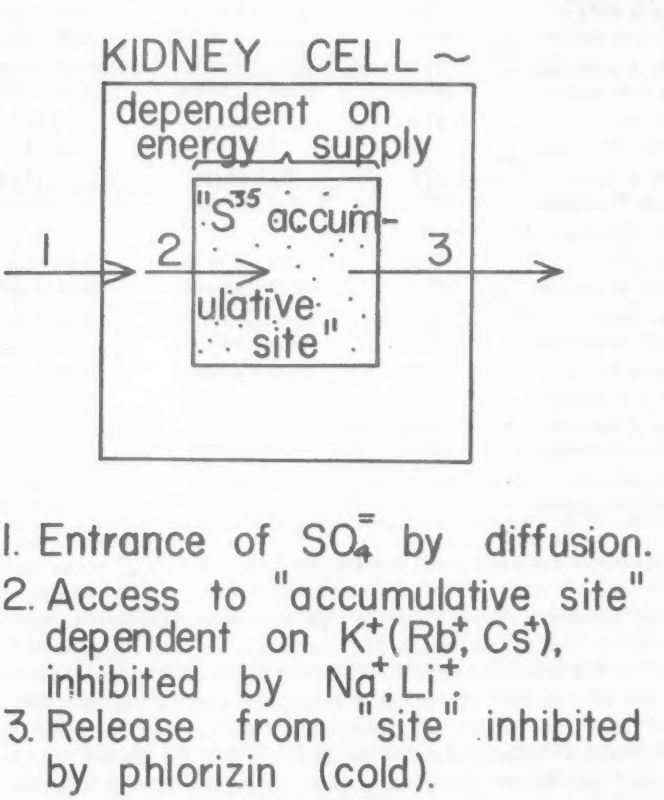

FIGURE 3. Scheme summarizing one of the possible hypotheses to account for the differential effects of cations and phlorizin on virtual tissue/medium S^{35} gradients of kidney cortex incubated at 38° C.

hypothetical schemes to account for the effect of these agents on net accumulation of S^{35} at 38° C. Additional work is planned to assess the effects of the alkali metal cations upon respiration and ionic patterns of renal tissue under the conditions of the experiment, with special reference to phlorizin action and variations related to temperature.

Summary

Radiosulfate accumulation by kidney cortex *in vitro* is enhanced by K^+, Rb^+, and Cs^+ and inhibited by Na^+ and Li^+ at 38° C. The relatively lower tissue/medium gradients developed at 0° C. are higher in the presence of Li^+ and Na^+ than when the incubation medium contains the other alkali metal cations. The inhibiting effects of Li^+ and Na^+ on S^{35} uptake at 38° C. are reversible to a large extent. Phlorizin enhances S^{35} uptake at 38° C. and inhibits it at 0° C., regardless of the species of cation present. These findings provide a basis for speculation as to possible mechanisms of renal S^{35} uptake *in vitro*.

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DIVISION OF NUCLEAR PHYSICS

ENERGY PRODUCTION FROM NUCLEAR REACTIONS*

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It is interesting to note that almost the entire crust of the earth is essentially in chemical equilibrium and consequently is useless as a source of chemical energy. The exceptions consist of deposits of past life which, together with present life, provide the energy we need. According to the first law of thermodynamics, the life process of manufacturing energy sources from stable compounds requires energy. The ultimate source of this energy is the sun which, as we now know, is powered by the nuclear burning of hydrogen into helium. Man's new-found ability to tap the nuclear energy reservoirs promises him for the first time a full material independence from his ancestral life, and may perhaps be regarded as a (material) coming of age.

The aims of this paper[1] are to review some basic facts of nuclei and nuclear reactions, leading to the conclusion that energy from nuclear reactions is best sought in the fusion of light elements and in the fission of heavy elements; to present some of the requirements and difficulties of controlled fusion reactions; and to discuss some of the energy consequences of the controlled release of fission energy.

Basic Review

The nuclei can all be represented by points on a two-dimensional grid. The coordinates most often used to identify a nucleus are Z, the atomic number or number of protons, and A, the mass number or total number of nucleons. The number of neutrons is then A-Z. A nucleus or atom is usually denoted by giving the chemical symbol with the atomic number as the left subscript and the mass number as the right superscript. Thus, ${}_1H^1$ denotes light hydrogen or the proton, and ${}_1H^2$ heavy hydrogen or the deuteron.

The natural nuclei, those found in nature, do not cover a large area on a Z,A plot. Instead, they fall within a very narrow band around the so-called stability line drawn in FIGURE 1. A number of the approximately 300 natural nuclei are unstable. Thus, all the elements with atomic number greater than 83, that of bismuth, are radioactive. Of all these heavy elements, only ${}_{92}U^{235}$, ${}_{92}U^{238}$, and ${}_{90}Th^{232}$ are present in large amounts, and they are the parents of the three natural radioactive families. There

*This paper was presented at a meeting of the Division on October 14, 1959.

The Division of Engineering held a meeting on October 16, 1959, at which Marvin M. Kessler of The M. W. Kellogg Company, New York, N. Y. delivered a paper entitled "Machine-Independent Computer Coding Systems." This paper will appear in a later edition of the *Transactions*.

also exist several natural radioactive nuclei that are not members of families. One such is K^{40}. Nuclei that are out of the stability band (above, below, or to the right along the dotted continuation in FIGURE 1) are unstable and transform spontaneously toward the stability band.

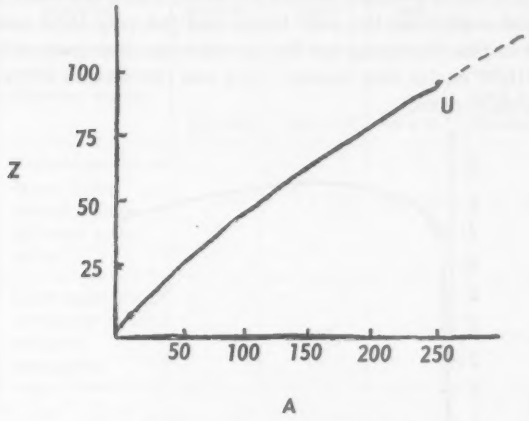

FIGURE 1. The stability line.

Questions of stability are related directly to considerations of energy. The energies involved in nuclear reactions may be measured directly by determining the energy released in an exothermic reaction or the energy input required for an endothermic reaction. The reaction energies may also be obtained from the results of mass spectroscopy, together with the mass energy equivalence relationship of Einstein. The ions of the atoms, when subjected to electric and magnetic fields, move differently depending on their masses. It is found from the resulting values of the mass ratios, as expected from Einstein's relation, that the mass of a nucleus is less than the sum of the masses of the individual protons and neutrons (by roughly 1 per cent). The energy equivalent of this mass difference is called the binding energy of the nucleus and is the energy required to separate the nucleus into its component protons and neutrons. It is also the energy released in the formation of the nucleus from free protons and neutrons. It may be noted that the binding energies of chemical compounds could bè obtained, in principle, by measuring the mass difference between the component atoms and the compound. Here, however, the differences are about 1 part in 10^9 and are too small for use.

The binding energy of a nucleus divided by the number of particles in that nucleus, the binding energy per particle, is the average energy by which a nucleon is bound to the nucleus and is a direct measure of the relative stability of the nucleus. The larger the binding energy per particle, the greater is the stability. A somewhat smoothed-out plot of the

measured binding energy per particle for the natural nuclei is presented in FIGURE 2. The energy unit used is the practical unit of nuclear physics, the million electron volt (mev). It is seen that the binding energy per particle is fairly constant at about 8 mev, but drops off for large A and for small A where periodic effects are seen. Thus, the least stable of all the natural nuclei are the very heavy and the very light ones. It is, then, natural to look for energy-producing reactions that involve the fusion of the very light nuclei into heavier ones and the fission of very heavy nuclei into lighter ones.

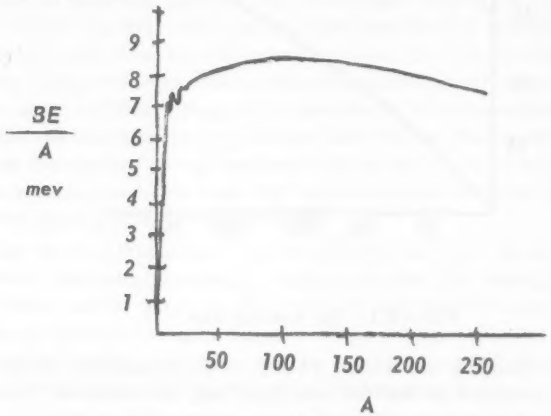

FIGURE 2. The binding energy per particle.

TABLE 1 presents a comparison of the energies involved in the fusion and fission nuclear reactions and in chemical and "physical" reactions by listing approximate values in some special cases. The ev unit is one electron volt and is the practical energy unit in atomic physics. The energy values for the chemical and nuclear reactions can be understood by making the following argument. In a chemical reaction, the important changes occur only in the outer electron orbits that have energies of roughly 10 ev. The energy involved in a reaction is expected to be given by the product of the number of outer electron orbits involved and the change in energy of these orbits. The energy should then be about 1 ev in order of magnitude, since the orbital energy change is only a fraction of the energy. In a nuclear reaction, the same argument leads to the product of the number of nucleons involved multiplied by the change in the binding energy per nucleon. For the listed reactions, this latter quantity is about 1 mev, and the number of particles is 2 for the fusion reaction and somewhat over 200 for the fission reaction.

Light Element Fusion and Thermonuclear Reactors

Some exothermic fusion reactions involving light elements are listed in

TABLE 1

ENERGY COMPARISON

Type of process	*Effect of process*	*Energy per process*	*Calories per gram*	*Calories per mole*	*Case*
Nuclear	Rearrangement of nucleons to form different nuclei	2 mev	23×10^9	46×10^9	Fusion of a neutron and a proton to form a deuteron
		200 mev	20×10^9	46×10^{11}	Fission of uranium
Chemical	Rearrangement of atoms (outer orbits) to form different molecules	1 ev	1×10^3	26×10^3	$C + O \rightarrow CO$
Physical	Rearrangement of molecules to form different structures	0.06 ev	80	1.4×10^3	Melting of ice

TABLE 2, together with their energy release values. These reactions are called fusion reactions because they result in the formation of a nucleus heavier than either of the original nuclei. Except for the two reactions using tritium, H^3, they all involve only natural nuclei. Tritium is unstable, with a half life of approximately 10 years, and must therefore be made.

In view of the listed reactions involving the hydrogen isotopes and in view of the general principle that anything that can happen does happen, why does water not burn with a nuclear fire? The answer lies in the rate of the reaction. The same question and the same answer apply to the burning of a wooden desk or, for that matter, a steel desk. The rate is

TABLE 2

SOME EXOTHERMIC FUSION REACTIONS FOR LIGHT ELEMENTS

$$_1H^1 + {}_1H^1 \longrightarrow {}_1H^2 + e^+ + \nu + 0.4 \text{ mev} \quad (\nu = \text{neutrino})$$

$$_1H^1 + {}_1H^2 \longrightarrow {}_2He^3 + \gamma + 6.2 \quad (\gamma = \text{photon})$$

$$_1H^1 + {}_3L_1^7 \longrightarrow 2\,{}_2He^4 + 17.3$$

$$_1H^2 + {}_1H^2 \longrightarrow {}_2He^3 + n + 3.3$$

$$_1H^2 + {}_1H^2 \longrightarrow {}_1H^3 + {}_1H^1 + 4.0$$

$$_1H^2 + {}_1H^3 \longrightarrow {}_2He^4 + n + 17.6$$

$$_1H^3 + {}_1H^3 \longrightarrow {}_2He^4 + 2n + 11.4$$

$$_2He^4 + {}_5B^{10} \longrightarrow {}_6C^{13} + {}_1H^1 + 4.0$$

determined by the number of collisions that take place between the reactant particles and by the fraction of the collisions that result in the occurrence of the reaction rather than in a simple scattering of the reactant particles. For the reactions in question, essentially no collisions occur in water because of the electrostatic repulsion between the nuclei. This Coulomb barrier may be overcome by speeding up the nuclei to sufficiently large energies. It may be thought that successful fusion power should be obtained by bombarding a target of water with a beam of hydrogen atoms that have been accelerated to energies sufficient to overcome the barrier (these are only 1 mev in order of magnitude). Due to the extremely small sizes of the nuclei, however, the projectile nucleus loses most of its energy by causing ionization before finding itself traveling head-on to a target nucleus, by which time it no longer has the required energy. The result, in using energies in the mev range, is that only one projectile particle among thousands causes the desired reaction. The energy gain is then only a very small fraction of the energy supplied to the accelerated beam and is much smaller than the energy loss due to inefficiencies in the accelerating process.

To cause a useful number of collisions requires not only the speeding up of the nuclei, but also the elimination of the energy loss to the electrons. Both of these conditions are met by raising the whole reacting mass to high temperatures (kindling), which is the first goal of the thermonuclear machines.

The criteria for choosing the material used in the machines are low Coulomb barrier and high probability of reaction per collision. The first criterion calls for the use of hydrogen because it has the smallest atomic number. The order of choice would be the reactions involving tritium and tritium, deuterium and tritium, deuterium and deuterium, proton and deuterium, and proton and proton. The last reaction involves only the abundant isotope of hydrogen, but is eliminated by the second criterion. The same objection applies, although not as strongly, to the proton deuterium reaction. Although tritium can be made in quantity in fission reactors, the major light element fuel is deuterium, of which there exists 1 atom in every 7000 atoms of hydrogen. Thus, the fusion reaction of major interest is that of deuterium burning with deuterium.

Successful fusion machines could supply us with as much energy as we can use for perhaps as long a time as the oceans remain or we depart. The problems involved in the kindling of a deuterium gas to the hundred-million-degree temperature range required for generating useful energy and in confining the hot gas for times sufficient for many collisions to occur are perhaps no more fantastic than some of the approaches made toward their solution. These problems and approaches are described by Amasa S. Bishop in his book *Project Sherwood*,[2] which makes fascinating reading.

Fission Reactors

In contrast, the first successful fission chain reaction took place as early as December 1942.

Although the only three abundant heavy elements, U^{235}, U^{238}, and Th^{232} undergo fission spontaneously, the rates are infinitesimally small for energy purposes. To obtain useful rates, the fission activation energy barrier must be overcome. This may be achieved by bombarding these nuclei with other nuclei. The use of natural nuclei is doomed to failure because of the Coulomb barrier that opposes collisions. There remains the only known neutral nucleus, the neutron that, however, is radioactive and transforms into a proton and an electron with a half life of approximately one quarter of an hour. The problem of supplying the neutrons is solved by the fission process itself, which yields an average of about 2½ neutrons per fission process. Thus, the chain reaction is possible whereby neutrons cause fissions that create neutrons causing further fissions, and so on.*

This possibility is realized only in U^{235}, in which the neutrons created in fission and the neutrons that may be retarded due to other processes can all cause further fissions in U^{235}. This situation is described by saying that U^{235} is thermally fissionable; that is, it can be made to undergo fission even with neutrons that have slowed down to speeds at which they are in thermal equilibrium with the surrounding matter. In U^{238} and Th^{232}, the binding energy of an additional neutron is about 1 mev less than the activation energy barrier. As a consequence, only neutrons of kinetic energy greater than 1 mev cause fissions, and the chain reaction cannot be sustained in these elements.

Fission reactors may be made, using natural uranium that contains 1 part U^{235} to 139 parts U^{238}, or uranium enriched in U^{235}. Heat, the major product of a fission reactor, may in part be converted into electricity by use of a heat engine (usually steam). There exist sufficient deposits of comparatively easily mined uranium to fill the world's energy need for many decades, if only the energy from fission in U^{235} is counted. This may be multiplied into many centuries by the use of breeding, and may be multiplied again by a large factor by use of all the uranium and thorium in the earth's crust.

Breeding involves the design of reactors in which more thermally fissionable atoms are made than are used. This is accomplished initially by mixing U^{238} or Th^{232} with U^{235} in reactors that convert the "fertile" isotopes U^{238} and Th^{232} into the thermally fissionable isotopes $_{94}Pu^{239}$ and U^{233}** by means of the following sequence of reactions.

*It should be pointed out that the natural death rate of neutrons is of no concern in reactors. In matter of ordinary density, the neutron is certain to be absorbed by a nucleus in a fraction of a second.

**These isotopes are radioactive with half lives of many thousands of years.

$$n + {}_{92}U^{238} \longrightarrow {}_{92}U^{239} + \gamma \qquad n + {}_{90}Th^{232} \longrightarrow {}_{90}Th^{233} + \gamma$$

$${}_{92}U^{239} \longrightarrow {}_{93}Np^{239} + e^- + \nu \qquad {}_{90}Th^{233} \longrightarrow {}_{91}Pa^{233} + e^- + \nu$$

$${}_{93}Np^{239} \longrightarrow {}_{94}Pu^{239} + e^- + \nu \qquad {}_{91}Pa^{233} \longrightarrow {}_{92}U^{233} + e^- + \nu$$

where γ, e, and ν stand for a gamma ray, a beta ray, and a neutrino. The newly made U^{233} and Pu^{239} may then be used to reproduce themselves in the same way.

The major drawback of fission power today is cost. The efficient design of present-day coal-burning power plants results in a somewhat lower cost per kilowatt hour. A discussion of atomic energy in the United States is presented in *Atoms for Power*, edited by Philip C. Jessup.[3]

One of the main advantages of fission energy over chemical energy is the vastly greater energy content achieved per pound of fuel. One application for which this advantage is of prime importance is the supply of heat or electricity to remote places such as stations in the Arctic and Antarctic regions, or even perhaps future stations on the moon or on artificial satellites. A similar application is the supply of propulsive power to vehicles traveling in out-of-the-way places. A good off-the-earth example is the use of a fission reactor to supply electricity to operate the ion drive of an interplanetary vessel. A second application of this advantage of fission energy is to increase the range of various earth-going vessels. For example, it would certainly be a comfort to the passengers and an aid to the crew if the airplane had the ability to travel several times around the world on one fuel loading. In the case of the submarine, the key advantage of fission energy is not the greater energy content that leads to a longer range, but rather the fact that the fission reaction may proceed while the submarine is submerged and cut off from the oxygen supply that is necessary for chemical propulsion.

Fission reactors produce large amounts of radioactive materials, some of which may be used as small sources of heat or in nuclear batteries. One type of battery consists of a central heat source connected to an outer radiator by thermocouples. A second type consists of a central rod containing a beta ray emitter separated from an outer cylindrical collector by an insulating medium. A very high voltage develops between the inner and outer electrodes. A third type incorporates a mixture of radioactive material and a phosphor in a central region sandwiched between a pair of photoelectric layers.

One of the major disadvantages of fission energy is the production of harmful radiations and of radioactive wastes that continue to emit harmful radiations. As a consequence, any reactor that is to produce power in the vicinity of people must be shielded. The thicknesses of shield required are usually measured in feet of concrete. There seems to be no possibility of ever having a compact car, or even a present-day car, powered by a shielded fission reactor. By optimizing the shield design, it is apparently

possible to use fission reactors to power very large airplanes. Assuming that the reactor itself can operate in complete safety, there remains the possibility of widespread damage due to radioactivity in the event of a crash in which the reactor is ruptured. Such damage can perhaps be avoided by having these planes take off, fly, and land only over water.

In any case, the supply of coal and oil will one day become depleted. Fission energy will then assume the entire function of heat and electricity: (1) if fusion energy has not partly replaced them; (2) if solar energy has not partly replaced them; and (3) if we survive.

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DIVISION OF INSTRUMENTATION

THE USE OF AN ANALOGUE COMPUTER FOR MEASUREMENT OF RESPIRATORY DEPRESSION*

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To evaluate the respiratory effects of a drug, it is desirable to have a method that measures respiratory effects and does not confuse metabolic effects with them. Changes in the state of consciousness also profoundly influence respiration, and effects due to drowsiness or sleep should not be interpreted as drug effects. In the search for potent analgesics, it is desired to find those that are devoid of side effects. The respiratory depressant influence of narcotic drugs is one of their main side effects, and often this side effect parallels other undesirable ones. An ideal method of measuring respiratory stimulation or depression in man should be sensitive enough so that a dose-response curve can be obtained with ordinary doses of narcotics such as 5 and 10 mg. of morphine. Furthermore, the method should be sufficiently simple to make it feasible to do multiple determinations giving the time of peak effects and the duration of drug action.

We have devised a method that will assess automatically and accurately the respiratory effects of drugs. The control determinations are highly reproducible within the same individual from hour to hour and from day to day. The basic physiological considerations in the development of this method and a brief description of the analogue computer employed will be presented.

Physiological Considerations

One of the simplest methods to measure respiration is to record respiratory rate. However, this is very inexact, variable and not a good index of respiratory depression or stimulation. Shallow, rapid breathing may be less efficient than normal breathing and may actually indicate respiratory depression. Thus, if respiratory rate alone were analyzed, depressant effects might actually be misinterpreted as stimulatory

* This paper, illustrated with slides, was presented at a meeting of the Division on October 20, 1959.

The Division of Psychology held a meeting on October 19, 1959, at which Herman A. Witkin, Department of Psychiatry, New York State College of Medicine, Brooklyn, N.Y., delivered a paper entitled "Psychological Self-Consistency." This paper will appear in a later edition of the *Transactions*.

effects. However, more probably, significant effects may be overlooked with this insensitive method.

Another method would be to measure only respiratory minute volume (total volume of air exhaled in one minute). However, there are two major objections. First, measurements of minute volume alone include metabolic as well as respiratory effects. Second, there may be respiratory depression associated with elevated tissue carbon-dioxide levels and, at the same time, no appreciable change in minute volume. In other words, minute volume measurements are an insensitive index. If drugs decrease the metabolic rate (decreased carbon-dioxide production) and the percentage of carbon dioxide in exhaled air remains the same, the minute volume is decreased, since carbon-dioxide production is volume times concentration.

Another method of studying respiratory depression or stimulation has been to study the respiratory response of a patient to elevated concentrations of inhaled carbon dioxide.[1] The resting ventilation is measured, and then the ventilation is measured while the subject is breathing 2.5 and 5 per cent CO_2. These values before and after the drug are plotted, and the best fitting lines are drawn. A change in slope (FIGURE 1) is usually interpreted as a change in sensitivity of the

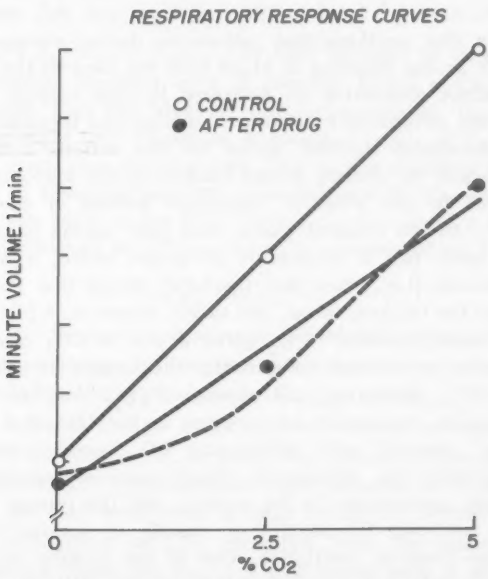

FIGURE 1. Control and 2 possible after-drug response curves. Values obtained while breathing room air and 2.5 per cent and 5 per cent CO_2. Dashed and solid after-drug lines illustrate two ways of interpreting data (see text).

respiratory center. In other words, after the drug there is less increase in ventilation for a given increase in CO_2, so that the slope is taken as a measure of respiratory center sensitivity. However, this method has some serious drawbacks if a continuous plot is not obtained. The three points measured may not define a true slope change. In fact, the dashed line in FIGURE 1 might just as well represent the true curve and, from our work, we have reason to believe that this is probably true. If this were true, the apparent change in slope would not be real, and changes in threshold would have been misinterpreted as changes in sensitivity. Since the after-drug curves are essentially straight lines at values of alveolar ventilation above 10 l./min. and bend toward the control curves somewhere below this value, the after-drug slope will also depend upon the concentrations of gases used.

In addition, there is the problem of equilibrium when the method of breathing mixtures of carbon dioxide is employed. The subject inhaling 5 per cent carbon dioxide, for instance, is seldom in carbon-dioxide equilibrium.[2] That is, there is a net accumulation of carbon dioxide in the tissues and bone due to the increased arterial and venous P_{CO_2}. This carbon-dioxide accumulation in the tissues means less net carbon-dioxide excretion, which in turn means less net ventilation. It takes at least 30 min. to approach equilibrium if elevated inspiratory P_{CO_2} levels are maintained.[2] Another source of error with this method stems from the fact that anything that influences tissue storage of carbon dioxide, such as the shunting of blood from the bone to the splanchnic area, will affect respiration as measured by this method. Also, this method will not differentiate between metabolic and respiratory effects.

The disadvantages outlined above for the various methods could be avoided largely by plotting minute volume against alveolar P_{CO_2}. We have preferred to plot alveolar ventilation instead of minute volume versus P_{CO_2}. Of the respired gases, only that coming in contact with the alveoli takes part in respiratory exchange; hence, it would appear better to measure this rather than the total volume that includes dead space air from the trachea, throat, and mouth. Moreover, a plot of alveolar ventilation versus alveolar P_{CO_2} gives a plot of CO_2 production and enables one to understand more readily the interrelationship between ventilation, CO_2 production, and alveolar P_{CO_2}. Another argument in favor of alveolar ventilation measurement is the fact that respiration is concerned primarily with maintaining CO_2 homeostasis, and this means controlling CO_2 elimination. Dead space represents an unproductive energy expenditure in the system, and the primary concern in determining how well the respiratory system is working is the final response: the alveolar ventilation. One of the primary advantages of plotting minute volume has been that it avoids the difficulty of calculating alveolar ventilation. However, now that the calculations can be done automatically, this advantage no longer exists. From the practical

point of view of evaluating the differences between drugs, both methods will yield essentially the same information.

One of the advantages of plotting ventilation versus P_{CO_2} is that it enables one to perceive readily the effects of inhaling carbon dioxide. It has been claimed that the respiratory center is more sensitive to the effect of narcotics when it is put under stress, but this is not the case. Narcotics, indeed, do produce a greater effect upon minute volume when a subject is inhaling 5 per cent carbon dioxide than when a subject is breathing room air, but this is not primarily the result of any change in the responsiveness of the respiratory center or respiratory control mechanism.

The reason for the increased effect of narcotics on ventilation when it is measured at elevated P_{CO_2} levels is apparent when one considers the equation for net carbon dioxide excretion. Let

$\dot{V}_A$ = alveolar ventilation in l./min.,

$\dot{V}_{CO_2}$ = carbon dioxide excretion in l./min.,

$P_{A_{CO_2}}$ = partial pressure of carbon dioxide in alveolar gas, and

P_B = barometric pressure at standard conditions 760 mm. Hg.

then: $\dot{V}_A (P_{A_{CO_2}}/P_B) = \dot{V}_{CO_2}$. The body attempts to maintain carbon-dioxide excretion constant even at elevated levels of CO_2. Nielsen's work[3] showed that the alveolar ventilation-alveolar P_{CO_2} response curve is a straight line. Therefore, at elevated values of P_{CO_2}, alveolar ventilation must also be elevated. The carbon-dioxide isopleth when the subject is breathing elevated concentrations of CO_2 asymptotically approaches the value of the CO_2 inhaled, in FIGURE 2 a P_{CO_2} of 40. Therefore, the formula for CO_2 excretion is represented by

$$\dot{V}_A (P_{A_{CO_2}} - P_{I_{CO_2}}/P_B) = \dot{V}_{CO_2},$$

where $P_{I_{CO_2}}$ is the partial pressure of the inhaled carbon dioxide. Since the alveolar ventilation is increased when breathing elevated levels of carbon dioxide, the difference between $P_{A_{CO_2}}$ and $P_{I_{CO_2}}$ will be small. Therefore, a small change in $P_{A_{CO_2}}$ will cause a large change in ventilation (*b* in FIGURE 2). A small drug-induced displacement in the alveolar ventilation P_{CO_2} response curve amounts to a small change in $P_{A_{CO_2}}$, and this must cause a large change in alveolar ventilation to maintain a constant net CO_2 excretion. Thus, the same small shift in the response curve (or $P_{A_{CO_2}}$) will cause only a minor change (*a* in FIGURE 2) in ventilation when the subject is rebreathing room air. This explains why the measurement of respiratory minute volume changes while the subject is breathing room air is a relatively insensitive

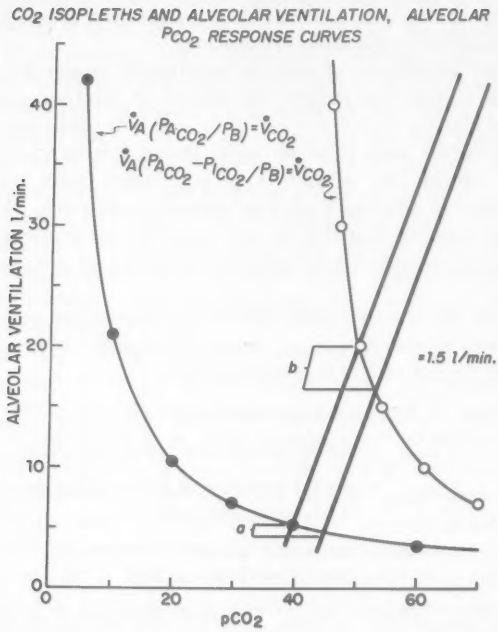

FIGURE 2. Idealized CO_2 isopleths for individual breathing room air (*solid circles*) and air containing 5.3 per cent CO_2 (*open circles*). Control and after-drug respiratory response curves also shown. Size of *a* compared to *b* illustrates why it is easier to detect effects of narcotics when breathing elevated tensions of carbon dioxide compared to room air (see text for explanation).

index of respiratory depression and why the change in ventilation measured at elevated inspired CO_2 levels is a more sensitive index.

In essence, in plotting alveolar ventilation versus alveolar P_{CO_2} we are describing the performance of a servo system. The respiratory minute volume under steady-state conditions is proportional to the work done to maintain respiratory homeostatic conditions: it is related to the output of the respiratory center. In other words, the respiratory center receives stimuli, compares them to some reference level, and sends impulses down the motor nerves so that the muscles may move air and maintain homeostasis. When the stimuli decrease, when ventilation is excessive, the output of the respiratory center decreases and vice versa. There is a feedback loop contained in the effect of ventilation on the blood and the effect of blood directly and indirectly on the respiratory center. Thus, the respiratory minute volume is related to the output of the respiratory center.

Ventilation is not necessarily proportional to the output of the respiratory center. If there is a decrease in compliance or an increase in

the airway resistance, ventilation will be decreased. Carbon-dioxide levels will be increased. This feedback will cause the output of the respiratory center to increase in order to maintain adequate ventilation. However, this ventilation is being attained by increased output of the respiratory center. Thus, because of varying frictional losses, the minute volume measurements will not always reflect accurately the output of the respiratory center.

In the plot of alveolar ventilation versus alveolar P_{CO_2} the alveolar P_{CO_2} may be thought of as corresponding to the feedback signal in a servo. The slope of the response curve is related to the gain of the respiratory center (sensitivity to CO_2) and disturbance factors (frictional losses – resistance, compliance). Parallel displacement of the response curve then represents a resetting of the servo reference level.

In developing a method to study respiration, we did not use the inhalation of fixed concentrations of carbon dioxide because of the problem of equilibrium and also because subjects cannot breathe comfortably at high levels of carbon dioxide for a prolonged time. We therefore used a modification of the method described by Eckenhoff *et al.*[4] whereby the subject rebreathes his endogenous carbon dioxide in a circle system and builds up the concentration of inhaled carbon dioxide gradually. It might be argued that the same problem of equilibrium arises here as is seen while breathing a fixed concentration of carbon dioxide. Actually it is different in that a true steady-state equilibrium is never reached. However, a dynamic balance is achieved in that there is a relationship between alveolar P_{CO_2} and brain tissue P_{CO_2} despite the fact that they are continually changing. Evidence for this stems from several facts: (1) alveolar ventilation versus alveolar P_{CO_2} plots are a straight line, and this agrees with the work of Nielsen,[3] who found a straight-line relationship under steady-state conditions; (2) extension of straight lines obtained from plots of rebreathing data during control runs are close to the room-air point and tend to indicate that the differential between alveolar and brain tissue P_{CO_2} is close to that present during steady-state conditions.[5]

We express respiratory depression or stimulation in terms of the shift of the alveolar ventilation curve. That is, at an alveolar ventilation of 15 l./min. the P_{CO_2} is determined for the control curve. This value is subtracted from the P_{CO_2} value at 15 l./min. for the 1 and 2 hours after drug determinations (line *A*, FIGURE 3). Thus, a positive number indicates respiratory depression, that is, a shift to the right, and a negative number indicates respiratory stimulation. We have found that, when the alveolar ventilation exceeds 10 l./min., the respiratory response curves tend to be parallel, so that valid comparisons may be made at or above this level. Another approach would be to measure ventilation at a constant P_{CO_2}. One pitfall in the employment of this technique is illustrated in FIGURE 3. The after-drug respiratory response

curves are not linear over their entire range. Usually, following drug, the curve assumes a slope parallel to the control curve above a specific alveolar P_{CO_2}. If measurements are carried out at a constant P_{CO_2} and expressed as changes in minute volume before and after drug (line *B*, FIGURE 3) and one is on a nonlinear portion of the response curve, then erroneous results may be obtained. It is possible that doubling the dose of a drug will produce little apparent effect. The straight position of the respiratory response curve could be extrapolated back so that measurements may be made at a constant P_{CO_2} but, in addition to the error due to extrapolation, one is faced with the interpretation of a negative value for ventilation.

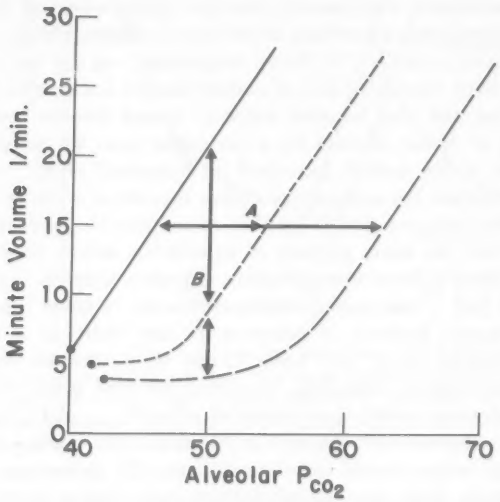

FIGURE 3. Possible respiratory response curves before and after drug. Solid line represents control curve. Small dashed line represents peak displacement following drug, and large dashed line represents peak displacement at double this dose.

Description of Computer System

The subject, fitted with a nose clip and rubber mouthpiece, breathes into the system through a one-way flutter valve (FIGURE 4). There are 2 three-way valves in the system, so that with both valves open the subject inhales room air through one leg of the system and exhales to the room through the other leg of the system. With both the valves closed and 6 l. of oxygen in the reservoir, the subject rebreathes in a closed-circle system, and the endogenous carbon dioxide rises. A polyethylene catheter is positioned in the one-way flutter valve so that its tip terminates at the level of the lips, and a continuous sample

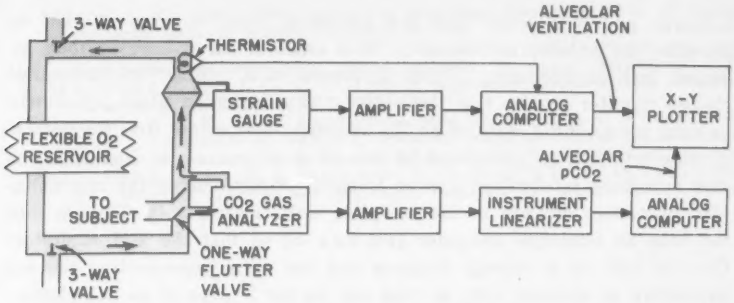

FIGURE 4. Block diagram of rebreathing circuits, sensing elements, amplifiers, computers, and XY plotter. Reproduced by permission from *Science*.[8]

of gas is drawn through the Listen-Becker LB-2 infrared gas analyzer at a rate of 500 ml./min. and returned into the system ahead of the the flow meter. Gas flow is sensed by measuring the pressure differential across a fine mesh screen.[6] In order to determine tidal volume, the output of the flow meter is amplified and fed into an analogue computer. In the computer (FIGURE 5) the flow is integrated to obtain tidal volume. The tidal volume is then corrected for barometric pressure and temperature, so that a volume of gas at standard pressure and at 37° C., saturated with water vapor (BTSP) is obtained. Since physiological dead space varies with tidal volume, the physiological dead space is determined from the cylindrical formula of Gray.[7] The tidal volume increases from the beginning to the end of a rebreathing run and, similarly, the dead space increases. The peak calculated dead space is held on a peak

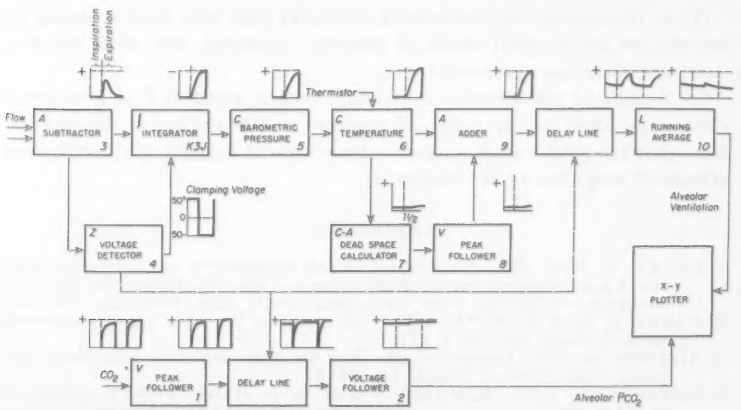

FIGURE 5. Block diagram of analogue computers for alveolar ventilation and alveolar P_{CO_2} calculation. Wave forms (output and polarity) of each stage are shown. Reproduced by permission from *Science*.[8]

follower and subtracted from the corrected tidal volume, so that an alveolar air volume is obtained. This alveolar air volume is then injected into an averaging circuit by means of a "delay" or sequential charge transfer line so that the alveolar ventilation in liters per minute is read out on the Y axis of an XY recorder. The output from the carbon-dioxide analyzer is processed by means of an instrument linearizer, so that a voltage is obtained that is linearly proportional to the concentration of carbon dioxide in the sampling cell (FIGURE 4). This is then fed into an analogue computer (FIGURE 5) so that the end expiratory CO_2 is held on a voltage follower and the value representing the end expiratory of alveolar CO_2 is read out on the X axis of an XY plotter. A detailed description of the circuits employed in the analogue computer has been published.[8]

This computer was deemed necessary since the plotting of the alveolar ventilation-alveolar P_{CO_2} response curves was tedious and time-consuming. In one study[9] more than 200 hours were expended in the plotting of response curves. This computer, in addition to providing rapid reduction of data, actually gives us more complete data in that each breath during rebreathing is represented on the continuous plot of the XY plotter. By observing the slope of the continuous plot of the XY plotter, subtle decreases in slope due to drowsiness on the part of the subject may be appreciated immediately.[10] Subjects are required to read a newspaper during the rebreathing runs. After their attention has been directed to this task, the slope of the response curve plot will return to the true slope.

Summary

(1) A respiratory response-curve computer that has proved useful in the evaluation of small doses of codeine, morphine, and other narcotic analgesics has been presented.

(2) A shift of the alveolar ventilation versus alveolar P_{CO_2} response curve was used as an index of respiratory stimulation or depression. Some physiological considerations relevant to the definition of respiratory effects of drugs have been discussed.

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DIVISION OF MYCOLOGY

ENZYMATIC AUTOINDUCTION AND THE HYPOTHESIS OF INTRACELLULAR PERMEABILITY BARRIERS IN *NEUROSPORA**

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It is my purpose in this paper to record in an organized manner some information pertinent to induced enzyme synthesis and enzyme function in *Neurospora*. Much of the information has been fragmentarily recorded in conjunction with experiments whose direct relevance to this subject has not been obvious.

All mutant strains of *Neurospora* thus far examined, that cannot carry out some single reaction subsequent to the synthesis of 5-dehydroshikimic acid (DHS) in the synthesis of the aromatic amino acids and *p*-aminobenzoic acid, accumulate protocatechuic acid (PCA) in their growth medium[1] (also S. R. Gross, unpublished observations). After exhaustion of the glucose or sucrose supplied, the PCA accumulated is quantitatively metabolized.[2]

It has been shown that:

(1) The PCA accumulated is derived from DHS by a dehydration catalyzed by dehydroshikimic acid dehydrase (DHS dehydrase).[3]**

(2) Protocatechuic acid oxidase (PCA oxidase) catalyzes the oxidation of PCA to β-carboxymuconic acid (β-CMA).[4,5]

(3) β-CMA is converted to β-ketoadipic acid (β-KA) *via* β-carboxy muconolactone or its corresponding hydroxy acid by enzymes referred to as lactonizing and delactonizing enzymes.[4]

(4) β-KA is split by a coenzyme A-dependent particulate associated enzyme yielding succinate and acetate.[6]

(5) Wild-type mycelia grown on synthetic medium with or without the aromatic amino acids contain at most an exceedingly small amount of PCA oxidase and no detectable DHS dehydrase despite the fact that DHS is an intermediate in the synthesis of the aromatic amino acids.[3,4]

(6) The aromatic amino acid-requiring strain Y7655 contains relatively large amounts of DHS dehydrase and PCA oxidase.[3,4]

(7) The synthesis of PCA oxidase can be induced in the wild-type strain by PCA or any one of a number of inducers added to the growth medium.[7]

* This paper, illustrated with slides, was presented at a meeting of the Division on October 23, 1959. The work described in this paper was supported in part by Research Grant C1267 from the National Cancer Institute, Public Health Service, Bethesda, Md.

** DHS also undergoes an extremely slow spontaneous conversion to PCA in the mildly acidic growth medium. The rate is too slow to account significantly for the accumulated PCA.

(8) It has not been feasible, thus far, to demonstrate the induced synthesis of DHS dehydrase in the wild-type strain.

(9) The enzymes catalyzing the conversion of β-CMA to β-KA are constitutive. There is no difference between the specific activity of these enzymes in extracts of the mutant and uninduced or induced wild-type strains.

The significant reactions described are illustrated in FIGURE 1.

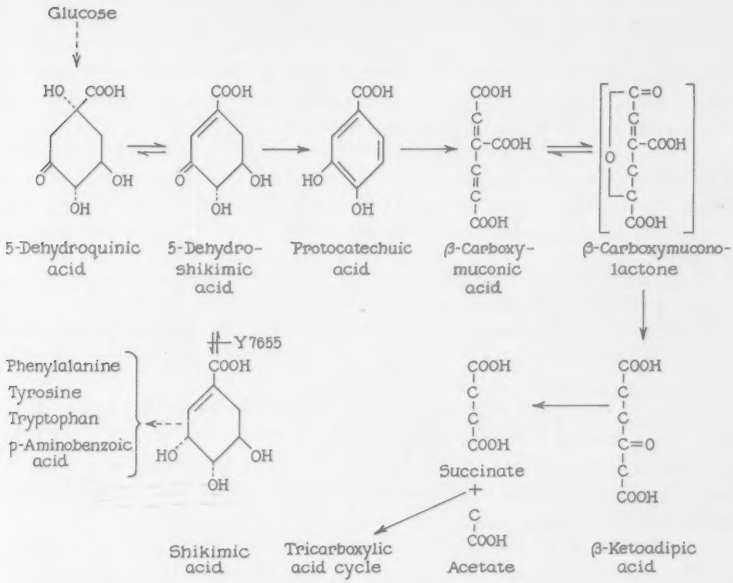

FIGURE 1. Reaction sequence discussed in body of text. The compound in brackets may not be a "true" intermediate, but isolated instead of the hydroxy derivative.

The synthesis of DHS dehydrase, it must be concluded, is induced in these mutant strains as a consequence of the interrupted conversion of DHS to the aromatic compounds. Indeed, a small amount of DHS was found to be accumulated in the growth medium of these strains [1] (also S. R. Gross, unpublished observations). The induction of PCA oxidase therefore follows the production of its substrate and inducer: PCA. This phenomenon, the induced synthesis of enzymes as a consequence of an endogenous metabolic alteration leading to the production or accumulation of an inducer, will be referred to as enzymatic autoinduction.

As already mentioned, although PCA is accumulated in considerable quantities during growth by the aromatic amino acid-requiring strain, Y7655, it apparently does not metabolize this compound until the sugar supplied as the carbon and energy source is depleted.

Pertinent information in support of this conclusion is:

(1) The amount of PCA accumulated is approximately equal to the amount of amino acids that must be supplied for equivalent growth (Gross, unpublished observations). This stoichiometric relationship suggests that the amount of PCA accumulated is equal to the amount of DHS that is normally produced as the precursor of the aromatic amino acids. Unless overproduction of PCA is uniquely balanced with PCA oxidation, it seems necessary to assume that PCA is not oxidized during the growth of the *Neurospora* mutant strain on sucrose or glucose.

(2) The activity of the enzymes involved in the synthesis of DHS from precursors is no higher in the mutant than in the wild-type strain as determined in extracts (Gross, unpublished observations). Therefore, no mechanism for PCA overproduction is evident.

(3) Wild-type mycelia induced to metabolize PCA will stop oxidizing PCA after a relatively short period of continued oxidation when glucose, sucrose, or certain other carbon sources are added to the growth medium. Oxidation of PCA resumes after the exhaustion of the added carbon source.[2]

That PCA is accumulated at all by the mutant is surprising in view of the following considerations:

(1) The specific activity of DHS dehydrase in the mutant is not more than that of PCA oxidase, although precise determinations are difficult because of the instability of DHS dehydrase.[3]

(2) The affinity of PCA oxidase for its substrate PCA is more than 100 times greater than that of DHS dehydrase for DHS. The K_m for DHS dehydrase is 6×10^{-4} M, while that of PCA oxidase has not been measurable by techniques that would detect a K_m of at least 5×10^{-6} M.[3] (Gross, unpublished observations).

(3) Glucose does not inhibit the activity of PCA oxidase in extracts.

(4) Induced wild-type mycelia are capable of metabolizing PCA added to the medium at a rate 3 to 5 times faster than the rate at which PCA is accumulated by the mutant strain. Nevertheless, the maximum specific activity of PCA oxidase found in wild-type extracts has been less than half that observed in extracts of the mutant.

The presentation of a specific biochemical hypothesis explaining the effect of glucose or its derivatives on PCA oxidase seems pointless because of the prevalence of such inhibitions in *Neurospora* as well as other organisms. The uptake of shikimic acid (Gross, unpublished observations) and inositol (A. Shatkin, personal communication) are both significantly inhibited by sucrose or glucose, or both.

The problem then is to account for the almost quantitative endogenous conversion of DHS to PCA by the mutant strain during its growth on glucose and sucrose and to reconcile its failure to metabolize PCA with the kinetic considerations listed above.

The carbon source inhibition of PCA metabolism in *Neurospora* is reminiscent of the "diauxie" displayed by certain bacteria that pref-

erentially utilize for growth one carbon source in a mixture until it is exhausted and resume growth at the expense of a second carbon source only after an extensive lag.[8] In *Escherichia coli* the inhibition by glucose of the induction of β-galactosidase has been attributed to the inhibition of an inducer concentrating mechanism.[9] It is quite possible that in the wild-type strain of *Neurospora* there are two mechanisms involved in the induction of PCA oxidase: one involves the uptake of PCA from the medium and its concentration in the cytoplasm; the other involves some mechanism controlling the availability of substrate at the site of enzyme function. The apparent failure of the oxidation of the endogenously produced PCA in the mutant grown in the presence of glucose where the synthesis of PCA oxidase is maximal plus the complete inhibition of the oxidation of PCA by glucose when added to preinduced wild-type mycelia suggest that: (1) at least one mechanism other than one responsible for transport of PCA across the cell membrane must operate, and (2) an inhibitory effect of glucose or a glucose derivative is exerted within the interior of the cell.

A rather vague but heuristic hypothesis accounting for these observations can be proposed. DHS dehydrase and PCA oxidase might be spatially separated in the cell in such a way that PCA cannot reach PCA oxidase in the presence of a derivative or metabolic product of glucose. One must assume that PCA oxidase is located on or in some structure bounded by a surface the properties of which can be altered in the presence of certain endogenous compounds. Although this concept seems reasonable, it should be noted that it has not yet been possible to demonstrate the association of PCA oxidase with any known cell particulate fraction. The complexity of the fine structure of the cytoplasm of most cells, however, is suggestive of protein organization other than that represented by the cell particulates (the mitochondria and microsomes) for which methods of isolation have been perfected.

The fact that wild-type mycelia do not contain a significant amount of DHS dehydrase even though DHS is a normal intermediate in the synthesis of the aromatic amino acids suggests the existence of a distinctive compartment for DHS dehydrase. Entrance of the substrate into this compartment seems not to be inhibited by a glucose derivative since essentially all of the DHS accumulated is converted to PCA by the mutant. It is not immediately evident, however, whether the concentration of DHS within wild-type mycelia ever reaches a level during normal growth sufficient to induce DHS dehydrase synthesis.

The hypothesis of intracellular compartments bounded by permeability or fixation barriers is not a new one in the sense that the existence of intracellular discontinuous metabolic pools has been well recognized. The examination of competition between various substances for entrance into endogenous pools, as apparently illustrated by the carbon-source effect, might be a useful approach to the differentiation between the study of intracellular compartments.

The notion of intracellular sites of fixation and/or concentration is presented to account for situations in which one can be confident that cell membrane permeability does not play a limiting role in enzyme function. Its pertinence to the extensive information on the *trans* cell membrane transport of inorganic ions and certain organic compounds is not immediately obvious. However, it might have some value in the interpretation of the apparent selectivity for certain organic compounds demonstrated by cells in which pinocytosis has been proposed as a mechanism whereby metabolites might enter the cell. Pinocytosis by its very nature offers no mechanism for the apparent exclusion of compounds entering in the same "gulp." Selective fixation within discontinuous intracellular compartments combined with outward transport or diffusion might be an experimentally useful way of looking at the problem.

Finally, it should be pointed out that enzymatic autoinduction is probably only one of many regulatory mechanisms determining the plasticity of the enzymatic complement of cells and tissues. Its observation in *Neurospora* was dependent upon the existence of a genetically determined loss of a specific enzyme activity and the consequent accumulation of enzyme inducers and substrates. Thus, the products of a biosynthetic series of reactions are shunted *via* two catabolic reactions not previously employed to any significant extent into a constitutive path of energy-yielding reactions (FIGURE 1).

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DIVISION OF ANTHROPOLOGY

DOWRY AND INHERITANCE IN MODERN GREECE*

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The farmers in the village of Vasilika, Boeotia, raise cotton and tobacco for sale. They also grow enough wheat to supply bread for their own families and for the shepherds they employ, and enough grapes to keep their households supplied with wine. The land on which these crops are grown, the machinery used to cultivate them, the houses in which the villagers live, and the money saved from the sale of the cash crops constitute the main forms of property for each village family. The success with which this property is handled largely determines the honor and prestige accorded each family by the villagers, and it contributes to the degree of pride a husband and wife and their children have in themselves.

What is considered success? The essential family obligation is to maintain a ratio between property and children such as to enable each child, when the property is divided into substantially equal shares among all the children, to maintain in his turn and for his family a decent standard of living. In other words, the villagers think of the elementary family as a kin group that conserves, accumulates, and transmits property from one generation to the next. The transmission of wealth is an important part of this trio of obligations. Property owned by a husband and wife who have no children to inherit it gives little satisfaction even when it means that the couple can maintain a relatively high standard of living.

Clearly, neither the forms in which wealth is reckoned nor the goals of the family with respect to it are unusual; they are certainly familiar to European rural societies. The occasion for this paper is the fact that the particular mechanisms by which property is inherited in Vasilika, with the social and cultural context in which they occur, have ramifications that seem especially worthy of note.

The patrimony is transferred to sons by inheritance or by gifts *inter vivos*, and to daughters by dowry. However, there is a strongly felt moral obligation to give marriageable daughters first lien on the property. The result is that the process for providing dowries for daughters and the patterns for the ultimate disposition of dowry properties have wide economic and social ramifications, some of which will be discussed here.

By Greek law and village custom each child is entitled to an equal share of property. While his children are still young, a farmer calculates approximately what he must give his daughters for dowries and what will be left for his sons to share. Sometimes it is obvious that the father's

*This paper, illustrated with slides, was presented at a meeting of the Division on October 26, 1959.

holdings, when distributed after his death to his sons in equal shares, will not be sufficient to support each of them. In such cases, one or more of the sons will be educated or trained for a nonfarming occupation. A son so trained is deemed to be drawing on his share of the patrimony. A family not only conserves its fields by educating its extra sons, but also gains added prestige by having a son in a white-collar civil service position or in a profession. The prestige of town occupations is so great that sometimes even an only son who is a promising student may be sent to continue his education in the gymnasium. The nearest gymnasium to Vasilika is approximately fifteen miles away, and some of the village boys go even further. The attendance of a son at one of these schools entails a cash outlay for room, board, and appropriate clothing. These expenses must be paid out of income from the land. In addition, although this has become less important with increasing mechanization, the absence of the son at school deprives the family of the value of his services either on the farm or as a hired laborer on other properties. Thus, the villagers seem to feel that a son educated away from home receives, in the course of the years, the equivalent of all or a major part of his prospective inheritance. The situation is considered the same even with less prestige-giving types of training or education, such as apprenticeship to a barber or a tailor.

In the meantime, the farmer has other calls on whatever monies he can save after providing for the ordinary subsistence needs of his family. There is first the desire to have as much cash as possible to supplement the land he will give for his daughters' dowries. The more gold sovereigns he has, the less land he must part with and, therefore, the more will be saved for his farming sons. Second, his daughters need money to supply part of the materials necessary for their trousseaus. The selling of cotton and tobacco, each season, is therefore an occasion for deciding whether some cash is to be spent on yet another article of the trousseau. The father and his sons are also always constantly aware of the ultimate need to relinquish some of the land and some of the laboriously collected savings when the girls marry.

The burdens of fathers with daughters are not relieved by procuring well-dowered wives for their sons, contrary to what those familiar with dowry systems in other parts of the world might expect. If it were possible to use the incoming dowry of a son to provide the property needed for the marriage of a daughter, the social and economic consequences of the Greek dowry system would be entirely different. Alternatively, if the Greek villager could avoid an actual transfer of property by having his son and his daughter marry the daughter and son of another farmer, as sometimes occurs in India, again the consequences would be different. Neither of these two alternatives is possible in Greek society. The fact is that the dowry, in every case, is considered property held in trust for the children of the newly married couple. It is therefore part of the de-

scending patrimony of the new family and not part of the ascending or collateral patrimony of the groom. The bride's dowry can be used neither by the groom's father, nor by the groom himself, for the benefit of the groom's sisters. Indeed, the marriage contract may permit all or part of the wife's dowry to remain the property of the wife herself. Even when the groom has control or supervision over the dowry properties – this is the customary arrangement of the villages, whatever the provisions of the formal contract – he thinks of this property as separate from that which he has already inherited or will inherit from his father. The former is *próika yinekós*, woman's dowry, the latter is *patrikó*, paternal inheritance. As you walk around Vasilika with a villager, he will describe one field as having been part of the dowry his mother brought his father, another as land that he purchased with money drawn from his wife's dowry. A third and fourth plot, no longer his, were given respectively by his father as part of his sister's dowry, and by himself as part of his eldest daughter's when she married. The villager's remaining fields, apart from any that he may have bought or obtained by exchange in an ordinary real estate transaction, he calls *patrikó*, paternal, for he reckons as *patrikó* any land that was part of his grandfather's holdings, regardless of its original source. Thus, it takes at least two generations for dower land to be incorporated into the patrimony of the male line into which it comes. The dowry is therefore a mechanism by which property is transmitted from a woman to her children with a period of intermediate administration by her husband. With the dowry, the inheritance system is a bilateral one; a man receives property through the ancestors of both his father and his mother.

Once the dowry agreement has been made and the marriage has taken place, the actual disposition of the types of property that comprise the dowry has further consequences in the Greek countryside. The ultimate disposition is, in turn, related to the social framework in which the dowry system operates. Vasilika may be defined in Murdock's terms as a *patrideme*, a community in which there is more than one separate and distinct kin group related through males; one in which men bring their wives from other communities and expect to send their daughters and sisters to be married elsewhere. Village exogamy is not a moral ideal, but is the most frequent arrangement, partly because a large proportion of the two hundred and twenty inhabitants of Vasilika are too closely related through the patrilineal line to avoid the incest prohibitions of the Greek Orthodox Church, and also because one is likely to be able to make more advantageous marriages in other communities.

Therefore, let us consider the disposition of the dowry of a Vasilika girl who marries a farmer's son in Elatia, several miles away. The girl, of course, goes to live in Elatia, where she and her husband are entitled to a room in his father's house. In this room, the couple and their subsequent children sleep. Her trousseau supplies bedding and the personal touch for the room. In addition. the young couple is entitled to support

from the joint efforts of the husband and his parents and siblings on the farm. The bride's dowry lands remain in Vasilika. Farming them requires either that her husband travel from Elatia to Vasilika to work the land himself, or that he engage one of his affinal relatives to work the land on shares. As he grows older, our Elatia farmer may prefer to exchange his wife's *príka* lands in Vasilika for some nearer his own village. The exchange might be arranged with a Vasilika man who brought his wife from Elatia or its vicinity. If the transaction requires additional payment, the cash may be drawn from the money part of the dowry that our Elatia farmer received with his Vasilika bride. Finally, if no reasonable exchange is practicable, the inconvenience of scattered land holdings may lead the Elatia farmer to sell the Vasilika dowry lands in order to purchase other land in or near his home.

A farmer son-in-law may also use the cash from his wife's dowry for capital outlays calculated to enhance the value of his children's patrimony. Therefore, he may buy major farm equipment, or additional land, or beehives, none of which will be merged with the assets of his paternal household, although the income drawn from the dowry-derived properties may be.

Let us now turn to the distribution of dowry properties that occurs when a village girl marries a townsman instead of a farmer, either in one of the provincial centers or in Athens. Here custom calls for a dowry that may include some land but comprises, as its principal element, a house in the town that must be habitable, although not necessarily complete, before the marriage can actually take place. The completion of the house remains the obligation of the father of the bride and may ultimately require the sale of some of his land to provide the necessary cash. Where a village girl marries an artisan or small tradesman living in a town, all or part of her dowry lands may be sold to establish her husband in business or to enable him to expand his enterprise. In all these instances, whatever dower lands there are in the bride's village, although they may be temporarily worked by affinal relatives on shares, are for the most part ultimately sold out of the bride's family.

The combination of village exogamy, patrilocal residence, and land as part of the dowry has the effect, therefore, of stimulating land transfers in the Boeotian countryside. Since some portion of each man's inherited lands, if he has any daughters, will pass into the hands of a different bilateral kin group, that is, a different elementary family, there is no opportunity to develop kin-associated holdings that will have the same boundaries for more than one generation. A son cannot expect to be farming exactly the same plots his father farmed, and he is even likely to own some lands in a village in which his father was entirely unknown. Indeed, a farmer cannot even expect, in the course of his own lifetime, to continue to farm the same land that constituted his holdings when he began as the head of a new elementary family. This situation

effectively prevents the strengthening of the patrilineal lineage as a significant kin group in spite of a pattern of patrilocal residence.

It is very likely that the Boeotian situation also precludes the development of strong sentiments with respect to particular plots of land and may therefore account for, or at least reinforce, the instrumental attitude toward property characteristic of the Boeotian villages. Land is neither loved nor revered, nor is it considered an extension of oneself. It has value as a source of income; cash is needed for savings and dowries as well as for subsistence; therefore, new agricultural techniques which obviously improve crop yields are welcomed and easily absorbed into the farming practices of the community. Furthermore, if land is merely a source of income, other means of earning a living can be in direct competition with it. As I have already indicated, the fact is that in Vasilika nonfarming occupations in the towns have more prestige than farming, and merchandising, skilled trades, or semiskilled work such as driving a taxi are acceptable alternatives.

The late Robert Redfield discussed the apparently contrasting attitudes toward land of northern and eastern European rural peoples as compared to those of Mediterranean peasantries. In the north and east, land has personal and mystical associations, and the industrious pursuit of agriculture has strong moral value in and of itself. On the other hand, Redfield suggests that the Mediterranean people tend toward the instrumental view of land and the preference for town occupations just described for modern Boeotia (Redfield, 1956). Redfield seeks an explanation for this difference in the relative wealth of farmers in the two areas, and also in the ancient Greek tradition of city-dwelling ultimately diffused throughout the Mediterranean area.

If we take our Greek community as an example of the Mediterranean group, it may be useful to compare it with a northern farming tradition, that of the Irish. In the Irish countryside, money is the only form the marriage portion takes. It is given by the bride's father to the groom's father; thus, the dowering of a daughter does not diminish the land holdings that the father of the bride must pass on intact to one of his sons. The result is a strong identification of a patrilineal line with a particular plot of land (Arensberg and Kimball, 1940). That this kin identification with a particular plot is of intense importance to the Irish farmer may perhaps be shown most clearly by what happens in an Irish country family that has no male heirs. Here, the incoming son-in-law is expected to pay a money-bride price substantially in excess of the value of his bride's estate. The purpose of this excess payment, according to Arensberg and Kimball "... may be ... to overcome the anomaly felt at the reversal of the usual roles of sons and daughters." Perhaps Arensberg and Kimball's point may gain greater significance if viewed somewhat differently. I suggest that it is not so much the reversal of male and female roles that calls for the extra payment as the fact that an incoming son-in-law will ultimately change the patrilineal identity of the estate.

By contrast, a Greek incoming son-in-law is almost always a poor man whose own inheritance would be too small to support a family. When we questioned the villagers on whether such a son-in-law brings with him the equivalent of a bride price, the villagers looked surprised and said that if he had anything to bring, he would not be moving into his wife's household. In other words, the Greek incoming son-in-law has low social prestige because he is felt to be entering his wife's household in the role of a needed agricultural laborer, and the fact that the sons of the marriage will have his name is a secondary consideration. In the Irish countryside, the incoming son-in-law takes over the role of the proprietor of an estate that ultimately will be associated with his and not his wife's father's line, in a situation in which the identification of patrilineal lines with a given plot of land lasts for generations and is presumably a source of prestige and pride.

This comparison, although limited in scope, suggests that there is a connection between dowry and inheritance systems and attitudes toward land and land tenure. It would appear that, as in Greece, the bestowal of dowries representing an equal share of the inheritance for women, given at least partly in the form of fields, may well contribute to a practical, utilitarian attitude toward land simply because it prevents the development of unilaterally owned permanent estates and, instead, fosters land sales and exchanges. The converse would seem to be the case with communities such as those of rural Ireland, where a dowry is bestowed in the form of money, leaving the patrilineal land intact. Further use of comparative material, with strict attention to the operational details of various systems of dowry and inheritance, might establish a definite correlation between these and the attitude toward land and land tenure among the rural peoples of Europe.

References

ARENSBERG, C. M. & S. T. KIMBALL. 1940. Family and Community in Ireland. :113-116. Harvard Univ. Press. Cambridge, Mass.

REDFIELD, R. 1956. Peasant Society and Culture. :105-117. Univ. Chicago Press. Chicago, Ill.

CONFERENCES HELD

SECTION OF BIOLOGICAL AND MEDICAL SCIENCES

FREEZING AND DRYING OF BIOLOGICAL MATERIALS

Thursday, October 1, and
Friday, October 2, 1959

Conference Chairman: Harold T. Meryman
Naval Medical Research Institute, Bethesda, Md.

THURSDAY, OCTOBER 1, 1959

PHYSICAL ASPECTS OF FREEZING

Session Chairman: Bruce Chalmers
Harvard University, Cambridge, Mass.

9:00 A.M. –
Greetings from the Academy - Robert L. Kroc, Chairman, Section of Biological and Medical Sciences, The New York Academy of Sciences, New York, N. Y.; Warner-Lambert Research Institute, Morris Plains, N. J.

"Introduction: General Principles of Freezing and Freezing Injury in Cellular Materials" - Harold T. Meryman, Naval Medical Research Institute, Bethesda, Md.

"Thermal Analysis of Eutectics in Freezing Solutions" - Louis R. Rey, L' École Normale Supérieure, Paris, France.

"Ice Crystal Formation in Biological Materials During Rapid Freezing" - John L. Stephenson, National Heart Institute, National Institutes of Health, Bethesda, Md.

"The Influence of Glycerol on Ice Propagation During Freezing" - C. V. Lusena, National Research Council, Ottawa, Canada.

FREEZING OF LIVING CELLS

Session Chairman: R. I. N. Greaves
Cambridge University, Cambridge, England

2:00 P.M. –
"Experimental Analysis of the Mechanism of Freezing of Aqueous Sol-

utions, Cells, and Tissues" - Basil J. Luyet, American Foundation for Biological Research, Madison, Wis.

"Freezing Injury of Plant Tissue" - J. Levitt, University of Missouri, Columbia, Mo.

"Factors Affecting the Erythrocyte During Rapid Freezing and Thawing" - A. P. Rinfret, Linde Company, Div. Union Carbide Corp., Tonawanda, N. Y.

"The Physical State Transition in The Freezing of Living Cells" - A. Cecil Taylor, The Rockefeller Institute, New York, N. Y.

"Physical Factors Implicated in the Death of Microorganisms at Subzero Temperatures" - Peter Mazur, Oak Ridge National Laboratory, Oak Ridge, Tenn.

FRIDAY, OCTOBER 2, 1959

FREEZE-DRYING

Session Chairman: Isadore Gersh
University of Chicago, Chicago, Ill.

9:00 A.M. –
"Theory and Practice of Freeze-Drying" - T. W. Rowe, Edwards High Vacuum, Ltd., Crawley, Sussex, England.

"Low Temperature Preparation Techniques for Biological Specimens Utilizing Liquid Helium II" - H. Fernandez-Moran, Massachusetts General Hospital, Boston, Mass.

"Radio Frequency Spectroscopy of Frozen Biological Material: Dielectric Heating and the Study of Bound Water" - A. L. Hopkins, Western Reserve University, Cleveland, Ohio.

2:00 P.M. –
"Preservation of Living Cells by Freeze-Drying" - R. I. N. Greaves, Cambridge University, Cambridge, England.

Robert L. Kroc
Chairman

Charles Noback
Vice-Chairman

THE METABOLISM OF ORAL TISSUES

Thursday, October 15,
Friday, October 16, and
Saturday, October 17, 1959

Conference Chairman: Philip Person
Veterans Administration Hospital, Brooklyn, N.Y.,
Rutgers University, New Brunswick, N.J.

THURSDAY, OCTOBER 15, 1959

PERSPECTIVES ON ORAL TISSUE METABOLISM

Session Chairman: William H. Sebrell
Institute of Nutritional Sciences,
Columbia University, New York, N.Y.

9:00 A.M.-
Greetings from the Academy - Robert L. Kroc, Chairman, Section of Biological and Medical Sciences, The New York Academy of Sciences, New York, N.Y.; Warner-Lambert Research Institute, Morris Plains, N.J.

Opening Remarks - Reidar F. Sognnaes, Harvard School of Dental Medicine, Boston, Mass.

"Some Observations on the Evolution of Oral Tissues" - Philip Person, Veterans Administration Hospital, Brooklyn, N.Y.; Rutgers University, New Brunswick, N.J.

"Oral Structures: Genetically and Anthropologically Considered" - Wilton M. Krogman, Philadelphia Center for Research in Child Growth; Graduate School of Medicine, University of Pennsylvania, Philadelphia, Pa.

"Prenatal Influences upon Oral Structures" - Josef Warkany and Harold Kalter, Children's Hospital Research Foundation and University of Cincinnati College of Medicine, Cincinnati, Ohio.

"Experimental Animal Studies of Oral Tissue Responses to Nutritional and Metabolic Variables" - James H. Shaw, Harvard School of Dental Medicine, Boston, Mass.

"Human Oral Tissue Changes in Diseases of Nutrition and Metabolism" - Herbert Pollack, Mt. Sinai Hospital, New York, N. Y.; Post-Graduate School of Medicine, New York University, New York, N. Y.

"Clinical Observations on the Modification of Human Oral Tissue Metabolism by Local Intraoral Factors" - D. Walter Cohen, University of Pennsylvania School of Dentistry, Phiiadelphia, Pa.; and Henry M. Goldman, Boston University School of Medicine, Boston, Mass.

Open Discussion.

TOOTH TISSUES

Session Chairman: Gerrit Bevelander
College of Dentistry and The Graduate School,
New York University, New York, N.Y.

2:00 P.M. –
"Interrelationship of Tooth Composition, Body Fluids, Diet, and Caries Susceptibility" - Albert E. Sobel, The Jewish Hospital of Brooklyn, Brooklyn, N. Y.

"Inorganic and Organic Components of Tooth Structure" - Finn Brudevold, Lucille T. Steadman, and Frank A. Smith, Forsyth Dental Infirmary, Boston, Mass., and University of Rochester, Rochester, N. Y.

"Electron Microscope Observations on the Fine Structure of Teeth" - David B. Scott, National Institute of Dental Research, Public Health Service, Bethesda, Md.

"X-Ray Diffraction Studies of Tooth Structure" - Otto R. Trautz, New York University College of Dentistry and The Graduate School, New York, N. Y.

"Disturbances in Tooth Formation" - Seymour J. Kreshover, National Institute of Dental Research, Public Health Service, Bethesda, Md.

"Dental Caries and Host Metabolism" - Julian D. Boyd, Veterans Administration Hospital, Huntington, W. Va.

FRIDAY, OCTOBER 16, 1959

THE SALIVARY GLANDS

Session Chairman: Leon H. Schneyer
University of Alabama Medical Center,
Birmingham, Ala.

9:00 A.M.-
"Studies of Salivary Gland Proteases" - Leo M. Sreebny, University of Washington School of Dentistry, Seattle, Wash.

"Regulation of Salivary Gland Amylase Activity" - Leon H. Schneyer and Charlotte A. Schneyer, University of Alabama Medical Center, Birmingham, Ala.

"Oxidative Metabolism of Salivary Glands" - Rose Mary Schneider, Veterans Administration Hospital, Brooklyn, N.Y.; and Philip Person, Veterans Administration Hospital, Brooklyn, N.Y. and Rutgers University, New Brunswick, N.J.

"Iodide Metabolism of Salivary Glands" - N. B. Myant, Medical Research Council, London, England.

"Endocrine Influences upon the Salivary Glands" - William G. Shafer, University of Indiana School of Dentistry, Indianapolis, Ind.

"Parotin: A Hormone of the Parotid Gland" - Yosoji Ito, Faculty of Pharmaceutical Sciences, Tokyo University, Tokyo, Japan.

Open Discussion

THE SALIVARY GLANDS (continued)

Session Chairman: James A. English
Office of Naval Research, Medicine and Dentistry Branch,
Washington, D. C.

2:00 P.M.-
"Salivary Apoerythein" - Ernest Beerstecher, University of Texas School of Dentistry, Houston, Tex.

"A Nerve Growth-Promoting Effect of Mouse Salivary Glands" - Rita Levi-Montalcini and Stanley Cohen, Washington University, St. Louis, Mo.

"Effects of Radiation upon the Salivary Glands" - James A. English, Office of Naval Research, Medicine and Dentistry Branch, Washington, D. C.

THE TONGUE AND ORAL MUCOSA

Session Chairman: George Stein
School of Dental and Oral Surgery, Columbia University
and Montefiore Hospital, New York, N.Y.

3:30 P.M.-
"Glycogen Metabolism in Normal and Abnormal Human Buccal Mucosa" - David Weisberger, Harvard School of Dental Medicine, Boston, Mass.

"Electron Microscope Studies of Keratinization" - John T. Albright, Harvard School of Dental Medicine, Boston, Mass.

"Experimental Animal Studies of Tongue Changes in Nutritional and Metabolic Disease" - Dimitri Afonsky, Department of Dental Research, University of Rochester, Rochester, N. Y.

"The Effects of Induced Multivitamin Deficiencies on the Human Tongue" - George Stein, School of Dental and Oral Surgery, Columbia University and Montefiore Hospital, New York, N.Y.; Herta Spencer, Division of Neoplastic Diseases, Montefiore Hospital, New York, N.Y.

SATURDAY, OCTOBER 17, 1959

THE PERIODONTAL TISSUES

Session Chairman: Marvin S. Burstone
National Institute of Dental Research,
Bethesda, Md.

9:00 A.M.-
"Some Aspects of Collagen Biosynthesis" - Bernard S. Gould, Massachusetts Institute of Technology, Cambridge, Mass.

"A Theory of Connective Tissue Behavior: Its Implications in Periodontal Disease" - Milton B. Engel, University of Illinois College of Dentistry, Chicago, Ill.

"Relations Between the Metabolism and Structure of Bone" - Daniel M. Laskin, University of Illinois College of Dentistry, Chicago, Ill.

"Histochemical Study of Enzymatic Processes in Teeth and Bone" - Marvin S. Burstone, National Institute of Dental Research, Public Health Service, Bethesda, Md.

"The Sequence of Events in Osteogenesis as Studied in Polyethylene Tubes" - W. J. Linghorne, Banting and Best Department of Medical Research, University of Toronto, Ont., Canada.

"Hydrolytic Enzyme Metabolism in Periodontal Tissues" - Vincent Lisanti, Tufts University School of Dental Medicine, Boston, Mass.

Open Discussion

THE PERIODONTAL TISSUES (continued)

Session Chairman: William J. Linghorne
Banting and Best Department of Medical Research,
University of Toronto, Toronto, Ont., Canada

2:00 P.M.-
"Bacterial Mechanisms in Periodontal Disease" - John B. Macdonald.

Ronald J. Gibbons, and Sigmund S. Socransky, Forsyth Dental Infirmary and Harvard School of Medicine, Boston, Mass.

"Effects of Protein and Amino Acid Deficiency upon Periodontal Structures" - Lucien A. Bavetta, University of Southern California School of Dentistry, Los Angeles, Calif.

"Oxidative Metabolism of Gingiva" - Bertram Eichel, Tufts University School of Dental Medicine, Boston, Mass.

"Interaction between Local and Systemic Factors upon the Genesis of Periodontal Lesions" - S. Sigmund Stahl, New York University College of Dentistry, New York, N.Y.

Open Discussion

Robert L. Kroc
Chairman

Charles Noback
Vice-Chairman

DIVISION OF MATHEMATICS

NUMERICAL PROPERTIES OF FUNCTIONS OF MORE THAN ONE INDEPENDENT VARIABLE

Thursday, October 8,
Friday, October 9 and
Saturday, October 10, 1959

Conference Chairman: Henry C. Thacher, Jr.
Argonne National Laboratory, Lemont, Ill.

THURSDAY, OCTOBER 8, 1959

ANALYTICAL FOUNDATIONS

Session Chairman: Henry C. Thacher, Jr.
Argonne National Laboratory, Lemont, Ill.

9:30 A.M. –
Introduction to the Conference - Henry C. Thacher, Jr., Argonne National Laboratory, Lemont, Ill.

"Approximation by Spherical Summability" - Salomon Bochner, Princeton University, Princeton, N. J.

Session Chairman: M. M. Andrews,
Air Force Office of Scientific Research,
Air Research & Development Command, Washington, D. C.

2:00 P.M. –
"Variation Diminishing Approximations" - I. J. Schoenberg, University of Pennsylvania, Philadelphia, Pa.

"Uniqueness Theorems for Holomorphic Functions" - J. J. Kohn, Brandeis University, Waltham, Mass.

"Functional Analysis as an Aid to Evaluating Remainders" - Arthur Sard, Queens College, New York, N. Y.

FRIDAY, OCTOBER 9, 1959

NUMERICAL METHODS

Session Chairman: Gertrude Blanch,
Aeronautical Research Laboratory, Wright Air Development Center,
Wright-Patterson Air Force Base, Ohio

9:30 A.M. –
"Derivation of Interpolation Formulas in Several Independent Variables" - Henry C. Thacher, Jr., Argonne National Laboratory, Lemont, Ill.

"Quadrature Methods for Functions of More than One Variable" - Arthur Stroud, University of Wisconsin, Madison, Wis.

Session Chairman: L. M. Milne-Thomson,
Mathematics Research Center, United States Army,
University of Wisconsin, Madison, Wis.

2:00 P.M. –
"Fitting Empirical Data" - George E. P. Box, Princeton University, Princeton, N. J.

"Solving Systems of Nonlinear Equations" - L. R. Turner, Lewis Flight Propulsion Laboratories, N. A. S. A., Cleveland, Ohio.

"Power Series Solution, by Machine, of a Nonlinear Problem in Two-Dimensional Fluid Flow" - R. D. Richtmyer, AEC Computing and Applied Mathematics Center, New York University, New York, N. Y.

SATURDAY, OCTOBER 10, 1959

RELATED PROBLEMS

Session Chairman: Knox T. Millsaps,
Air Force Missile Development Center,
Holloman Air Force Base, N. Mex.

9:30 A.M. –
"Numerical Aspects of Partial Differential Equations" - Fritz John, Institute for Mathematical Sciences, New York University, New York, N.Y.

"Monte Carlo Methods for Solving Multivariate Problems" - J. M. Hammersley, United Kingdom Atomic Energy Research Establishment, Harwell, England.

Session Chairman: Henry C. Thacher, Jr.
Argonne National Laboratory, Lemont, Ill.

2:00 P.M. –
"Mathematical Programming" - H. W. Kuhn, Princeton University, Princeton, N. J.

"Summary and Conclusions" - Session Chairmen

NEW MEMBERS

Elected June 15, 1959

LIFE MEMBERSHIP

Mazo, Milton, M.D., Pediatrics. Private Practice, Savannah, Ga.

SUSTAINING MEMBERSHIP

Lewisohn, Marjorie, M.D., Internal Medicine, Instructor in Medicine, Cornell University Medical School, New York, N.Y.

Pace, John W., M.D., Neurological Surgery. Private Practice, Fresno, Calif.

ACTIVE MEMBERSHIP

Aceto, Mario D., Ph.D., Pharmacology. Assistant Professor, University of Pittsburgh, Pittsburgh, Pa.

Alberti, Vincent S., M.D., Medicine. Private Practice, New York, N.Y.

Angeluscheff, Zhivko D., M.D., Hearing & Deafness. Head, Hearing & Speech Center, City Hospital, New York, N.Y.

Anthony, William A., M.D., Cardiology. Private Practice, Gastonia, N.C.

Apgar, Virginia, M.D., Obstetrics. Professor, Anesthesiology, Columbia University, New York, N.Y.

Bader, Eli, M.D., Psychiatry. Psychiatrist, Private Practice, Chicago, Ill.

Bassett, James G., M.D., Gastric Physiology. Clinical Associate Professor of Surgery, University of Pennsylvania, Philadelphia, Pa.

Beckhorn, Edward J., Ph.D., Microbiology. Assistant Director, Research, Wallerstein Company, Staten Island, N.Y.

Beiser, Max, M.D., Biological Sciences. Private Practice, New York, N.Y.

Bell, David M., Jr., M.D., Medicine. Private Practice, Canonsburg, Pa.

Bianchi, Mary Ann, B.A., Research Biologist. U.S. Government Laboratories, Westerly, R.I.

Blank, H. Robert, M.D., Psychoanalysis. Private Practice, White Plains, N.Y.

Block, Max, M.D., Medicine. Private Practice, Montclair, N.J.

Bonilla-Sosa, Salvador, M.D., Antibiotics. Pfizer Corporation, Havana, Cuba.

Bowers, Wesley Creveling, M.D., Otorhinolaryngology. Private Practice, New York, N.Y.

Bryer, Ben F., M.D., Gastrointestinal Physiology. Associate Visiting Surgeon, New York City Elmhurst Hospital, New York, N.Y.

Camarata, Samuel J., M.D., Thoracic and Cardiovascular Surgery. Private Practice, Santa Ana, Calif.

Caputto, Ranwel, M.D., Biochemistry. Head, Biochemistry Department, Oklahoma Medical Research Foundation, Oklahoma City, Okla.

Cella, Robert L., M.D., Medicine. Private Practice, Oakland, Calif.

Correll, James William, M.D., Neurological Surgery. Instructor, College of Physicians and Surgeons, Columbia University, New York, N.Y.

Cotter, Sister St. Virginia Marie, Ph.D., Zoology. Professor, Biology, Notre Dame College, Staten Island, N.Y.

Cuthbertson, Elizabeth M., M.D., Experimental Surgery. Assistant Director, Children's Hospital, San Francisco, Calif.

D'Amato, Gabriel A. P., M.D., Psychiatry. Assistant Clinical Professor, Medical College of Georgia, Augusta, Ga.

Davis, Oscar F., Ph.D., Psychiatry. Resident Physician, University of Illinois, Chicago, Ill.

Doscher, Nathan, Ph.D., Health Education. Supervisor of Student Teaching, Brooklyn College, Brooklyn, N.Y.

Edelsack, Edgar A., B.S., Nuclear Physics. Head, Scientific Department of Naval Research, San Francisco, Calif.

Eger, Sherman Alfred, M.D., General Surgery. Clinical Professor of Surgery, Jefferson Medical College, Philadelphia, Pa.

Eichler, Bernard B., M.D., Medicine. Instructor in Medicine, Columbia College of Physicians & Surgeons, New York, N.Y.

Esman, Morris, M.D., Internal Medicine. Chief, St. John's General Hospital, Pittsburgh, Pa.

Feder, Samuel L., M.D., Psychiatrist. Assistant Attending Psychiatrist, Mt. Sinai Hospital, New York, N.Y.

Feindel, William H., M.D., Nervous System. Professor, University of Saskatchewan, Saskatoon, Canada.

Franco, Robert, M.D., Surgery. Private Practice, Richland, Wash.

Friedman, Leonard, M.S., Biochemistry. Research Assistant, Rutgers University, New Brunswick, N.J.

Friedmann, Lawrence W., M.D., Peripheral Vascular Disease, Institute of Physical Medicine & Rehabilitation, New York, N.Y.

Fukuchi, Genichiro, Dr. Pharm., Pharmaceutical Chemistry. Director, Sankyo Company, Tokyo, Japan.

Garver, Kenneth L., M.D., Medicine. Instructor, Pediatrics, University of Pittsburgh, Pittsburgh, Pa.

Goldstein, Emanuel, M.D., Cardiology. Private Practice, New York, N.Y.

Gordon, Joseph, M.D., Chest. Private Practice, Albuquerque, N. Mex.

Greene, David G., M.D., Cardiopulmonary physiology. Professor, Clinical Research in Cardiovascular Disease, University of Buffalo, Buffalo, N.Y.

Haas, Gerhard J., Ph.D., Microbiology. Director, Research, Liebman Breweries, Inc., Brooklyn, N.Y.

Happe, Lawrence J., M.D., Medicine. Member Hospital Staff, Minneapolis, Minn.

Harloe, John P., M.D., Fibrinolysin. Private Practice, Charlotte, N.C.

Harvey, Irving W., M.D., Medicine. Private Practice, Dorchester, Mass.

Hawes, Roland R., D.M.D., Dentistry. Eastman Dental Dispensary, Rochester, N.Y.

Hollander, Edward E., M.D., Psychoanalysis. State University of New York, Brooklyn, N.Y.

Horowitz, Helen Barbara, M.D., Endocrinology. In charge of Diabetic Research, Montefiore Hospital, New York, N.Y.

Hurd, James B., M.D., Internal Medicine. Associate in Medicine, Northwestern Medical School, Chicago, Ill.

Jones, Ben C., Jr., M.D., Internal Medicine. Private Practice, Alexandria, Va.

Klein, Alexander, M.D., Cardiology. Private Practice, Whitestone, N.Y.

Klinger, John S., M.D., Medical Sciences. Associate Physician, West Jersey Hospital, Camden, N.J.

Kolker, Joseph, M.D., Psychoanalysis. Clinical Assistant Psychiatrist, Mt. Sinai Hospital, New York, N.Y.

Leake, Chauncey D., Ph.D., Physiology. Ohio State University, Columbus, Ohio.

Leiby, A. L., M.D., Dermatology. Private Practice, Akron, Ohio.

Leonard, Edwin W., Hair & Skin Research. Sared Laboratories, Inc., Columbia, S.C.

Leuzzi, Anthony P., M.D., Medicine. Private Practice, Yonkers, N.Y.

Lodwick, Gwilym S., M.D., Radiology. Professor, University of Missouri School of Medicine, Columbia, Mo.

Loeb, Roland Allan, M.D., Cytology. Associate Physician, Lancaster General Hospital, Lancaster, Pa.

Loewenfeld, Irene E., Ph.D., Neurophysiology. Assistant in Ophthalmology, Columbia University, New York, N.Y.

Lowen, Jack, B.S., Chemistry. Chemist, Needham, Mass.

McLaughlin, Eugene James, M.D., Cancer. Private Practice, Dayton, Ohio.

Marcus, Herbert R., M.D., Disorders of Thyroid. Associate Attending Physician, Beth Israel Hospital, New York, N.Y.

Nelson, Gordon G., M.D., Surgery. Chief, Surgical Services, Youngstown Hospital, Youngstown, Ohio.

Newberry, William B., Jr., M.D., Medicine. Western Reserve University, Cleveland, Ohio.

Noë, Alfons F., Dr.Ing., Enzymes. Research Director, Wallerstein Company, Staten Island, N.Y.

Ostashever, Alvin S., Ph.D., Biochemistry. Food Drug Research Laboratories, Maspeth, N.Y.

Pappas, Arthur M., M.D., Tissue Transplantation. Naval Medical School, Bethesda, Md.

Passaretti, Anthony V., M.D., Urology. St. Mary's Hospital, Troy, N.Y.

Pisani, Bernard J., M.D., Obstetrics. Director, Obstetrics & Gynecology, St. Vincent's Hospital, New York, N.Y.

Quinn, Edward L., M.D., Internal Medicine. Physician-in-charge, Henry Ford Hospital, Detroit, Mich.

Redner, Bernard, M.D., Medicine. Private Practice, Brooklyn, N.Y.

Rommer, J. Jay, M.D., Neuropsychiatry. Assistant Clinical Professor, New York University Medical College, New York, N.Y.

Rothman, Ronald M., B.A., Medicine. Medical Student, Albert Einstein College of Medicine, Bronx, N.Y.

Schwartz, Mortimer L., M.D., Cardiovascular. Assistant Professor of Clinical Medicine, Seton Hall College of Medicine, Jersey City, N.J.

Segard, Edwin S., M.D., Medical Surgery. Retired, New York, N.Y.

Siclari, Vincent, Biophysical Medical Electronics Instruments. Representative, Medical Division, Sanborn Company, Brooklyn, N.Y.

Siltzbach, Louis E., M.D., Internal Medicine. Associate Clinical Professor, Columbia University College of Physicians and Surgeons, New York, N.Y.

Skalko, Richard G., M.S., Physiology. Instructor in Biology, St. John's University, Brooklyn, N.Y.

Splitter, Samuel R., M.D., Psychosomatic Medicine. Private Practice, Hempstead, N.Y.

Stahman, Albert W., M.D., Internal Medicine. Assistant Attending Physician, Brooklyn Hospital, Brooklyn, N.Y.

Sullenberger, S. D., M.D., Radiation. Administrator of Sullenberger Clinic & Hospital, Dandridge, Tenn.

Sutherland, G. F., M.D., Medicine. Private Practice, Big Stone Gap, Va.

Tice, Walter Thomas, M.D., Internal Medicine. Private Practice, High Point, N.C.

Torghele, John R., M.D., Psychobiology. Psychiatrist, Hastings State Hospital, Hastings, Minn.

Tyler, Edward T., M.D., Reproductive Problems. Director, The Tyler Clinic, Los Angeles, Calif.

Walter, Albert M., M.D., Chemotherapy. Bacteriologist, Bakteriologisches Laboratorium, Wuppertal-Elberfeld, Germany.

White, Thomas Taylor, M.D., Breast Cancer. Clinical Assistant Professor of Surgery, University of Washington School of Medicine, Seattle, Wash.

Winter, Fred O., M.D., Medicine. Private Practice, New York, N.Y.

Yellin, Mattis, Ph.C., Geriatrics. Nursing Home Owner, Flushing, N.Y.

Zisowitz, Milton L., A.M., History of Science, Medicine. Lecturer in Medical Literature, Cornell University College of Medicine, New York, N.Y.

Zausner, Joseph, M.D., Diagnostic Radiology. Associate Professor, New York University Bellevue Medical Center, New York, N.Y.

STUDENT MEMBERSHIP

Nathanson, Milton E., B.S., Entomology. Graduate Student, Teaching Assistant, Brooklyn College, Brooklyn, N.Y.

Prutkin, Lawrence, B.S., Cytochemistry. Graduate Student in Biology, Brooklyn College, Brooklyn, N.Y.

Elected July 15, 1959

LIFE MEMBERSHIP

Jaslow, Robert I., M.D., Pediatrics. Clinical Director, Pennhurst State School (institution for the mentally retarded), Chambersburg, Pa.

ACTIVE MEMBERSHIP

Adelman, Jack A., M.D., Internal Medicine. Instructor in Medicine, University of Texas Post-Graduate Medical School, San Antonio, Texas.

Allin, Eardley S., M.D., Surgery. Instructor in Surgery, University of Alberta, Edmonton, Alta., Canada.

Audia, Marino L., M.D., Physiology. Assistant Professor in Physiology, University of Panama School of Medicine, Panama, Republic of Panama.

Bass, V. V., M.D., Gynecology. Private Practice, Saginaw, Mich.

Bernstein, Daniel S., M.D., Endocrinological Research. Instructor, Harvard Medical School, Boston, Mass.

Coles, Robert S., M.D., Ophthalmology. Assistant Clinical Professor, New York University-Bellevue Medical Center, New York, N.Y.

Desai, Rajendra G., Ph.D., Hematology. Stanford University School of Medicine, San Francisco, Calif.

Deutsch, Harry L., M.D., Medicine. Private Practice, Los Angeles, Calif.

Donoho, Charles Porter, M.D., General Surgery. Private Practice, Dallas, Texas.

Earle, Julius R., M.D., Medicine. Private Practice, Walhalla, S.C.

Evans, Silas M., M.D., Atherosclerogenesis. Private Practice, Milwaukee, Wis.

Falk, Alfred B., M.D., Dermatology. Assistant Professor, University of Illinois College of Medicine, Chicago, Ill.

Feldmann, Pauline, M.D., Psychology. Resident Physician, Hospital of the Daughters of Jacob, Bronx, N.Y.

Fisher, J. S., M.D., Pediatrics. Private Practice, New York, N.Y.

Fleischer, Ronald S., M.D., Internal Medicine. Private Practice, Hyattsville, Md.

Friedman, Jacob H., M.D., Clinical Psychiatry. Director, Neuropsychiatric Service, Fordham Hospital, Bronx, N.Y.

Gilmore, William E., M.D., General and Thoracic Surgery. Surgeon, Camden-Clark Memorial Hospital, Parkersburg, W. Va.

Goddard, David R., Ph.D., Plant Physiology. Kuemmerle Professor of Botany, University of Pennsylvania, Philadelphia, Pa.

Goolker, Paul, M.D., Psychiatry. Faculty, New York Psychosomatic Institute, New York, N.Y.

Goodfriend, Milton J., M.D., Enzymology. Attending Obstetrician and Gynecologist, Lebanon Hospital, New York, N.Y.

Graig, Frank A., M.D., Cancer and Isotopes. Montefiore Hospital, Bronx, N.Y.

Grossman, Gilbert, M.D., Medicine. Private Practice, New York, N.Y.

Haney, Catherine A., M.D., Pediatrics. The Permanente Medical Group, San Francisco, Calif.

Harward, Thomas G., M.D., Medicine. Private Practice, Needles, Calif.

Hawley, Donald K., M.D., Internal Medicine. Private Practice, Cincinnati, Ohio.

Heiden, Lionel M., M.D., Psychoanalysis. Private Practice, New York, N.Y.

Henderson, Theodore A., M.D., Medicine, Instructor in Medicine, University of Pennsylvania, Philadelphia, Pa.

Henson, Clifton W., M.D., Medicine. Associate Attending Surgeon, Polyclinic Hospital, New York, N.Y.

Hofmann, J. William, M.D., Gynecology. Professor Emeritus, Indiana University Medical Center, Indianapolis, Ind.

Holley, Edward B., M.D., Pediatrics. Private Practice, Sacramento, Calif.

Hough, Heloise Beekman, M.D., Medicine. Radiology, Goldwater Memorial Hospital, Welfare Island, New York, N.Y.

Hubly, James W., M.D., Surgery. Private Practice, Battle Creek, Mich.

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Jaffe, Leonard David, Sc.D., Metallurgy. Chief, Materials Section, California Institute of Technology, Pasadena, Calif.

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Revitch, Eugene, M.D., Neuropsychiatry. Psychiatrist, New Jersey State Diagnostic Center, Plainfield, N.J.

Reyes-Garcia, R., M.D., Medicine. Private Practice, Springfield, Mass.

Richards, Robert R., M.D., Surgery. Private Practice, Fairview Park, Ohio.

Robins, Alvin L., M.D., Neurology. Assistant Professor, New York University College of Medicine, New York, N.Y.

Rocco, Albert F., M.D., Radiology. Private Practice, Greenville, R.I.

Rogers, Wilson J. H., B.S., Chemist. Director of Research, Industrial Testing Laboratories, New York, N.Y.

Rossen, Ralph, M.D., Biology. Director, L. E. Phillips Psychobiological Research Division, Mount Sinai Hospital, Minneapolis, Minn.

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Smith, J. Lawrence, M.D., Clinical Surgery. Private Practice, Iowa Falls, Iowa.
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von Tischler, Joseph Arthur, M.D., General Medicine. Private Practice, Bronx, N.Y.
Vorys, Nichols, M.D., Obstetrics. Instructor, Department of Obstetrics and Gynecology, Ohio State University Medical School, Columbus, Ohio.
Watts, Edgar Alan, M.D., Radiobiology. Research Scientist, Columbia University Radiological Research Laboratory, New York, N.Y.
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Wiedeman, George H., M.D., Psychoanalysis. Private Practice, New York, N.Y.
Willinger, Hyman, M.D., Psychiatry. Private Practice, New York, N.Y.

STUDENT MEMBERSHIP

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Carter, Jerry J., M.S., Cytology. Graduate Student, Fordham University, New York, N.Y.
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Elected August 17, 1959

LIFE MEMBERSHIP

Lieu, Vi-Tuh, M.D., Human Pathology. Assistant Pathologist, Long Island College Hospital, Brooklyn, N.Y.

SUSTAINING MEMBERSHIP

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ACTIVE MEMBERSHIP

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Rogols, Saul, M.Sc., Bacteriology. Research Bacteriologist, Ohio State University Health Center, Columbus, Ohio.

Roncoroni, Mario, M.D., Biochemistry. Staff Member, Sanatorio Parque and Hospital, Rosario, Argentina.

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Rusan, Thomas E., M.D., Gynecology and Obstetrics. Private Practice, Webster Groves, Mo.

Sata, William K., M.D., Neurology. Private Practice, Seattle, Wash.

Savory, P. M. H., M.D., Medicine. Private Practice, New York, N.Y.

Schaffenburg, Carlos A., M.D., Endocrinology. Medical Adviser, Ciba Co., Ltd., Montreal, Canada.

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Shaulis, Frederick S., M.D., Medicine. Private Practice, New York, N.Y.

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Steptoe, Philip P., M.D., Medicine. Associate Professor, Georgetown University Medical School, Washington, D.C.

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Sugar, Samuel J., M.D., Internal Medicine. Assistant Professor, George Washington University, Washington, D.C.

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Woodruff, Benjamin, M.D., Internal Medicine. Private Practice, Tracy, Calif.

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Weaver, John C., M.D., Medicine. Private Practice, New York, N.Y.

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Williams, B. H., M.D., Pediatrics. Head, Department of Pediatrics, Scott & White Clinic, Temple, Texas.
Williams, Eugene W., M.D., Endocrinology. Private Practice, Dearborn, Mich.
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STUDENT MEMBERSHIP

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Elected September 15, 1959

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Braun-Falco, Otto, M.D., Dermatology. Johannes Gutenberg Universitat, Mainz, Germany.
Brücke, Franz, M.D., Pharmacology. Professor, University of Vienna, Vienna, Austria.
Carmichael, Hugh T., M.D., Psychiatry. Clinical Professor of Psychiatry, University of Illinois College of Medicine, Chicago, Ill.
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Fleischer, Werner Richard, M.D., Medicine. Staff Pathologist, Mercy Hospital, Janesville, Wis.
Froix, Cleo J. L., M.D., Surgery. Resident, Harlem Hospital, New York, N. Y.
Goldstine, Herman H., Ph.D., Mathematics. Resident Manager, IBM Research Center, Yorktown Heights, N.Y.
Goodman, Benjamin, B.S., Cancer Chemotherapy. Vice-President, Philadelphia Ampoule Laboratories, Inc., Philadelphia, Pa.
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Owen, Robert L., M.D., General Medicine. Private Practice, Bradford, Vt.
Palmer, Allan, M.D., Infertility and Gynecology. Private Practice, San Francisco, Calif.
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Reed, A. Palmer, M.D., Obstetrics and Gynecology. Private Practice, Los Angeles, Calif.
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Roth, Donald A., Ph.D., Metabolism. Chief, Metabolism, Endocrinology and Renal Diseases, Wood Veteran's Administration Center, Wood, Wis.
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Webb, John J., D.D.S., Dental Research. LCDR. Dental Corps, U.S. Navy, Bethesda, Md.
Whitaker, H. S., M.D., Biological and Medical Sciences. Medical Officer, U.S. Penitentiary, Atlanta, Ga.
Williams, Dwight, Ph.D., Chemistry. Director of Research, St. Louis, Mich.
Williams, John R., M.D., Neurosurgery. Consultant, Butterworth Hospital, Grand Rapids, Mich.
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Elected October 22, 1959

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Abom, Robert H., A.B., Metals. Director, Edgar C. Bain Laboratory for Fundamental Research, Monroeville, Pa.

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Bello, Jake, Ph.D., Protein Chemistry. Senior Research Associate, Protein Structure Project, Polytechnic Institute, Brooklyn, N.Y.

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